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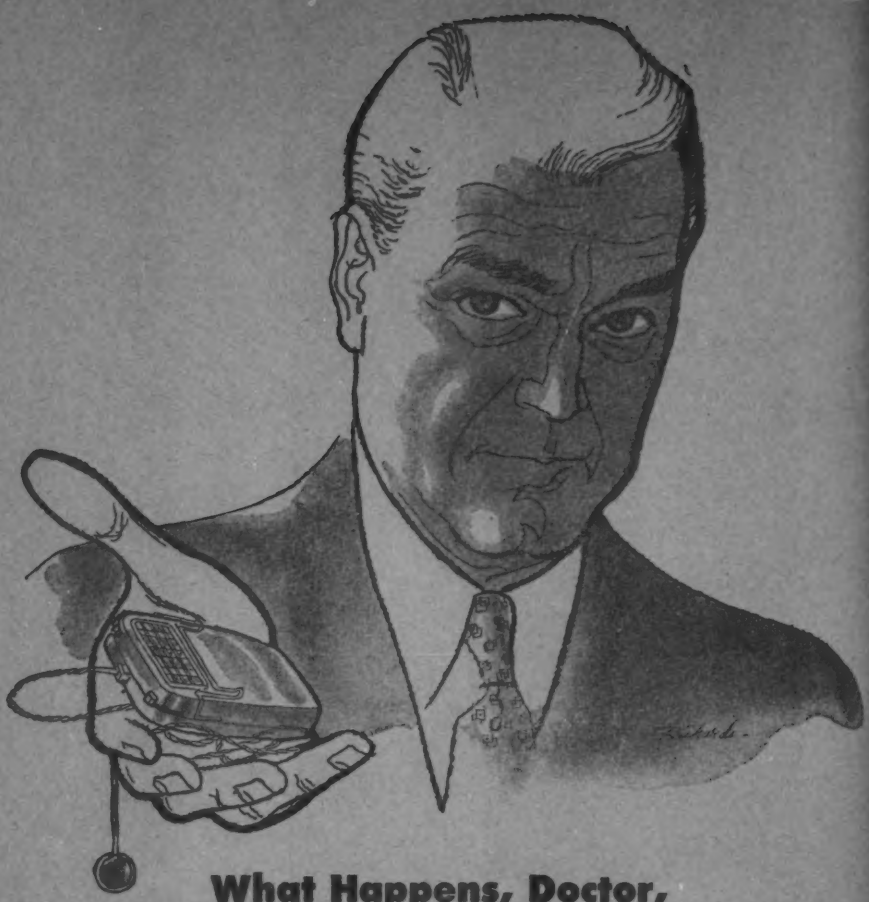

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THE ELECTROLYTES OF THE LABYRINTHINE FLUIDS.*

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The endolymph and perilymph of the inner ear have been the subject of speculation for many years. Theories concerning their origin and relation to each other still lack definite proof. Anatomical evidence points toward two fluids in separate compartments. Experimental evidence has been conflicting as to the permeability of Reissner's membrane and other parts of the membranous barrier. Little is known about their normal composition. From existing analytical data it appears that the labyrinthine fluids differ but slightly from spinal fluid and each other.

An extensive analysis of the pooled ear fluids from freshly killed sharks (Scolioidontus laticandus) was made by Kaieda[1] in 1930. There have been several attempts to repeat the analysis of fluids from living mammals. Table I gives values reported by previous investigators. Only those results from Kaieda's analysis that are pertinent to the present studies are included.

Kaieda[1] showed that shark perilymph had a slightly higher organic content than endolymph; endolymph had a higher in-

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organic content, as shown by its higher electrolyte concentration. Ledoux and his associates analyzed dog and cat labyrinthine fluids for a number of constituents. They observed small but consistent differences in the osmotic pressure of endolymph, perilymph, cerebrospinal fluid, and plasma.[2,3] Ledoux[4] reported that the protein content of cat perilymph is twice that of endolymph, and almost ten times that of spinal fluid. Waltner and Raymond[5] found much less protein in human perilymph (Meniere's disease). Szasz[6] gives a higher value

TABLE I.
DATA OF OTHERS ON ENDOLYMPH, PERILYMPH AND SPINAL FLUID.

	Animal	Perilymph	Endolymph	Spinal Fluid	Author
Viscosity (Relative to water)	Pigeon	1.7	2.9		Rossi[10]
Refractive Index					
$N_D^{17.5°}$	Dog	1.33515		1.33527	Szasz[6]
$N_D^{22.5°}$	Dog	1.3349	1.3347	1.3342	Ledoux[7]
$N_D^{22.°}$	Cat	1.33495	1.33455	1.33435	Ledoux[7]
Osmostic Pressure					
Relative to plasma	Cat	1.006	1.004	0.998	Ledoux[3]
NaCl equivalent in m.Eq./L	Cat	179	181	174	Aldred, Hallpike and Ledoux[9]
pH	Cat	7.87	7.82	7.45	Ledoux[9]
Electrolytes					
Sodium m.Eq./L	Shark	231	542	253	Kaieda[1]
Potassium m.Eq./L	Shark	14	24	14	Kaieda[1]
Chlorides m.Eq./L	Shark	245	464	231	Kaieda[1]
	Cat	152	158	151	Ledoux[8]
Protein mg. per cent	Cat	268	118	31	Ledoux[4]
	Human*	27		13	Waltner and Raymond[5]

* Meniere's cases. Average of three cases.

for the refractive index of spinal fluid compared to perilymph in the dog, while Ledoux[7] gives a lower value. Values given by Ledoux[4,8] for urea N, chlorides, and reducing substances show negligible differences in the three fluids. Waltner and Raymond[5] reported that a single sample of human perilymph had almost the same sodium concentration as spinal fluid.

Graf and Poretti[11] (1950) measured the rate of entry of radioactive Na into the labyrinthine fluids of the guinea pig. They found that the radioactive Na in the perilymph increased

from 20 per cent of the spinal fluid concentration at 37 minutes to 82 per cent at six hours. During this same interval, the concentration of the tracer in endolymph increased only from 5 per cent of the spinal fluid level to 9 per cent. There was little change in this ratio between one and six hours. The low initial levels in endolymph might indicate a slow exchange of the sodium in endolymph with perilymph or plasma; however, the failure of the level to rise appreciably after an hour suggests another possibility: that there may be a low total sodium concentration in endolymph.

To explore this possibility and to supplement the incomplete knowledge of labyrinthine fluid composition, the endolymph, perilymph, spinal fluid and plasma of the guinea pig have been analyzed for Na, K, Cl, and protein. Because of the changes which have been shown to occur in Na and K concentrations during propagation of the nerve impulse, these ions were of special concern. It has been found that endolymph differs strikingly from perilymph and spinal fluid in regard to Na and K.

SURGICAL TECHNIQUE.

Healthy, dark colored guinea pigs were used. They were of 250-350 gm. weight (6-8 weeks of age) with good hearing as shown by the pinna reflex. The animals were anesthetized with "Dial" (diallylbarbituric acid with urethane, Ciba), 0.5 ml. per kg., and 0.5 ml. of 1 per cent procaine.

Perilymph. The bulla was exposed and an opening made in it by dental drill. The tympanic membrane was carefully displaced from about half of its bony attachment in order that the lateral portion of the bulla, including the external ear canal, could be removed without undue tension on the ossicular chain. Both round and oval windows were then fully accessible. The round window membrane was cleaned of any tissue fluid by a wisp of cotton or small piece of gelfoam. The membrane was then pierced by a pyrex micropipette in a micromanipulator, and perilymph was withdrawn from the scala tympani of the cochlea. The pipette was made of 3 mm. tubing drawn down to 0.3 mm., 1 cm. from the tip, and 0.1 mm. at the tip. The pipette remained in the perilymphatic space for a minimal period (30-40 seconds) to avoid the possibility of contamination through the cochlear aqueduct.

Utricular Endolymph. The malleus and incus were next removed and the facial nerve unsheathed and displaced. The stapes was pulled up out of the oval window and the opening enlarged to give better visibility of the utricle. The vestibule was kept free of perilymph by intermittent suction at the round window. The utricle was pierced by a micropipette in a micromanipulator. As the endolymph was withdrawn, the utricle visibly collapsed. Although care was taken to avoid the capillaries of the round window membrane and utricle, this was not always possible. If more than a trace of blood was visible, the sample was discarded. The samples obtained ranged from 0.2 to 1 μl. (.0002 to .001 ml.).

Cochlear Endolymph. The cochlea was exposed as described above. In two animals (Nos. 89 and 98) the basilar membrane was pierced at the round window to enter the cochlear duct. The method has been described by Aldred, Hallpike and Ledoux[2] and by Graf and Poretti[11]. The vestibule and cochlea were cleared of perilymph. Constant suction, or a piece of gelfoam at the cochlear aqueduct was necessary to prevent the entry of spinal fluid. The technical difficulties could not be adequately overcome, and the method was abandoned, although a few valid specimens were obtained.

In other animals the cochlear duct was entered in the second or third turn through the stria vascularis. This was exposed by making a small hole in the bony cochlear wall with dental burr and steel pick. The round window was opened and the stapes loosened in the oval window to reduce the flow of perilymph through the artificial fenestra. A small piece of gelfoam was placed on either side of the hole. This appeared to absorb adequately the perilymph, and the outer layer of the stria vascularis seemed to be free of fluid. The stria vascularis was pierced by a small micropipette (40 microns outside diameter). A small amount of fluid immediately ran up by capillary attraction. Usually nothing more could be withdrawn by suction, and rarely could more than 0.4 μl. of fluid be obtained.

Cerebrospinal fluid was removed from the fourth ventricle. In some animals blood was removed from the heart for serum analysis.

All pipettes were cut short immediately upon withdrawal, sealed at both ends with paraffin or beeswax and refrigerated until surgery was completed. Most of the animals were perfused through the aorta with Heidenhain-Susa fixative, and the operated ear was removed, dehydrated and embedded in celloidin. Serial sections were stained with hematoxylin and eosin.

Sections from pigs which had furnished utricular endolymph showed that the medial wall of the utricle had been broken in only one animal of 22 examined. The ears from the remaining animals showed a large hole in the lateral wall of the utricle near the macula, where the pipette had entered. In three cases there was microscopic evidence of injury to the vestibular cochlear duct during the enlargement of the oval window. In four instances there was a small tear in the saccule at the point where it is attached to the utricle. As considerable pressure was sometimes necessary to pierce the utricular membrane, this tear was probably made as the pipette entered the utricle. In four cases sections of the vestibular cochlear duct were incomplete, due to faulty technique. The remainder showed the membranous labyrinth to be completely intact except for the hole made by the pipette.

Histological examination demonstrated less success in the collection of uncontaminated cochlear endolymph. On the basis of the examination of serial sections and the operative protocols, the animals were divided into three classifications according to the degree of confidence in the samples (Table IV). Data from one animal were discarded, because sections revealed puncture of Reissner's membrane.

ANALYTICAL TECHNIQUE.

Because of the limited amounts of fluid available, micromethods had to be developed or adapted specifically for the purpose. The special micropipettes, tubes and general techniques are those previously described for another purpose.[12] The reader is referred to this publication for details of pipetting, mixing, performing colorimetry, etc., on this scale.

The principles of the chloride method and necessary precautions have been published,[12] but the method has been re-

vised to provide increased precision. This is possible because of the greater amount of chloride present. The sodium method is adapted from an earlier procedure.[13] The K measurements were made with two different methods: in endolymph, which has a high K concentration, dipicrylamine[14] was used as a precipitation agent. In the other fluids precipitation was effected with chloroplatinic acid.[15] This latter reagent is more sensitive, but gives less precision at this microscale.

Chloride. To 0.2 μl. volumes of sample or standards (0.1, 0.12 and 0.14 *M* NaCl) were added 2.5 μl. 0.013 *M* $AgNO_3$ in 0.5 *N* HNO_3. After 30 minutes the samples were centrifuged, and a 2.0 μl. aliquot of supernatant fluid was *added to* 500 μl. of rhodanine reagent. This reagent is freshly prepared by mixing 4 ml. of 1 *N* H_2SO_4, 1 ml. of 10 per cent gum arabic, and 0.25 ml. of 50 mg. per cent solution of 5-(*p*-dimethylaminobenzylidine)—rhodanine (*p*-dimethylaminobenzalrhodanine, No. 2748, Distillation Products Industries, Rochester, N. Y.). After 30 minutes the samples were read at λ470 mμ. Replicate samples of inner ear fluids had a standard deviation of 0.7 millimoles per liter (0.6 per cent).

Serum was analyzed for chloride by a similar procedure. To 50 μl. of serum or standard (0.090, 0.10 and 0.11 *M* NaCl) were added 100 μl. 2.2 *N* HNO_3. After centrifuging, 50 μl. of the supernatant fluid was precipitated with 100 μl. of 0.020 *M* $AgNO_3$ in 0.1 HNO_3. Color was developed with 5 μl. of the supernatant fluid (obtained by centrifuging) added to 1 ml. of rhodanine reagent. A correction was made for the fact that only about 94 per cent of serum is water and that consequently, when serum is treated with two volumes of nitric acid, the chloride is diluted 2 per cent less than the chloride of standards.

Sodium. To 0.2 μl. volumes of sample, standards (0.025 and 0.150 *M* NaCl) or blank (water), in 50 mm. pyrex tubes of 2 mm. bore with pointed tips, were added 4 μl. of Barber and Kolthoff reagent,[16] which had been centrifuged before use. The samples were centrifuged after an hour, and a volume of supernatant fluid as close to 4 μl. as possible was removed without disturbing the precipitate. The precipitates were washed ("buzzed") with 4 μl. of a solution consisting of 20

per cent of Barber and Kolthoff reagent and 80 per cent glacial acetic acid. Exactly 4 μl. of the supernatant fluid was removed after centrifuging. (Throughout the precipitation and washing, it was necessary to avoid evaporation by keeping the tubes capped with Parafilm whenever possible). The precipitate was dissolved in 15 μl. water, and 10 μl. aliquots were added to either 500 μl. of color reagent (spinal fluid, perilymph, and 0.15 *M* standard) or to 200 μl. of color reagent (endolymph and .025 *M* standard). The color reagent was a fresh mixture of 25 ml. of 50 per cent acetic acid, 0.25 ml. of 10 per cent ascorbic acid, and 0.5 ml. of 25 per cent potassium ferrocyanide (fresh or frozen).[13] Readings were made after 10 to 60 minutes at λ470 mμ. The molar extinction coefficient, is about 12,000. The standard deviation for 0.2 μl. samples of perilymph and spinal fluid was 6 millimoles per liter, or 4 per cent. This could probably be improved by further study of precipitation and wash conditions.* The precision was, however, sufficient for the present purpose. The standard deviation with endolymph was about 1 millimole per liter or 6 per cent.

Serum sodium was determined in a similar manner, even though it might have been measured with a flame photometer, in order to use the same method for all fluids. Protein was removed from 50 μl. of serum by adding 5 μl. of 55 per cent trichloracetic acid and centrifuging. Sodium was precipitated from 1 μl. of supernatant fluid with 20 μl. of Barber and Kolthoff reagent. After centrifuging, 20 μl. of supernatant fluid were removed and the precipitates were washed with 20 μl. of glacial acetic acid. The precipitates were dissolved in 20 μl. of water, and color was developed by adding 5 μl. of the resultant solution to 1 ml. of color reagent. The standard deviation was 2.2 millimoles per liter or 1.5 per cent.

* A somewhat better procedure, used for some of the last samples, seemed to be the following: 0.2 μl. of sample was precipitated with 2 μl. of Barber and Kolthoff's reagent. After standing and centrifuging, 1.7 μl. of supernatant fluid were removed and the precipitate was washed with 2 μl. of glacial acetic acid. After centrifuging 2 μl. of supernatant fluid were removed. Subsequent steps were unchanged.

Potassium. To 0.2 μl. volumes of *endolymph* and standards (0.12 and 0.15 *M* KCl) were added 1.7 μl. of 0.03 *M* dipicrylamine in 0.033 *M* NaOH. After standing 30 minutes at room temperature the samples were centrifuged, and a 1 μl. aliquot of the supernatant fluid was added to 1 ml. of 0.005 *N* NaOH and the optical density was measured at λ430 mμ. No precipitation occurred with less than 6 mM K per liter (0.6 mM per liter during precipitation). The molar extinction coefficient is about 26,000 for *increments* of potassium. The standard deviation was 2 mM per liter with standards (1.5 per cent). Duplicate endolymph samples had a standard deviation of 8 mM per liter.

To 0.2 μl. samples of *perilymph* and *spinal fluid* and standards (.004, 0.006, and .008 *M* KCl) and blanks were added 1.6 μl. of 1 per cent H_2 $PtCl_6$ $.6H_2O$ in *n*-butanol. These were centrifuged after one hour at room temperature and 1.6 μl. of supernatant fluid were removed. The precipitates were washed once with 10 μl. of *n*-butanol containing 0.02 per cent chloroplatinic acid and once with 10 μl. of *n*-butanol alone. (The chloroplatinic acid could probably be omitted from the first wash.) The precipitates were dissolved in 40 μl. of 0.1 *N* HCl, and the optical density was measured at λ259 mμ. The molar extinction coefficient was about 12,000 (referred to K).

Protein was measured with 0.6 μl. aliquots by a published colorimetric method.[17]

RESULTS.

A total of 350 analyses were performed on serum, spinal fluid, and labyrinthine fluids from 53 guinea pigs. Only rarely was it possible to make all measurements on fluids from the same animal.

The most striking finding is that Na and K concentrations are almost reversed in utricular endolymph and perilymph (Tables II and III). The endolymph K concentration is 30 times that of perilymph and spinal fluid, whereas the Na concentration is 10 times higher in perilymph than in endolymph.* The concentration of the chloride ion is about 10

* It was possible with the flame photometer to confirm the high K concentration in a few larger endolymph samples, although the precision of measurement was less than with the chemical methods. The values found were 109, 137, 144, and 132 millimoles per liter in endolymph, compared to 7 and 5 millimoles per liter in two large samples (1 μl.) of perilymph.

TABLE II.

CHLORIDE, SODIUM AND POTASSIUM OF SERUM, SPINAL FLUID, PERILYMPH AND UTRICULAR ENDOLYMPH.

All values expressed as milliequivalents per liter. The endolymph for one animal had Na and K values of 58 and 76 m.Eq. per liter, without obvious explanation. These data have been omitted from the averages.

	Serum	Serum ultrafiltrate (calculated*)	Spinal fluid	Peri-lymph	Endolymph (utricle)
Chloride	93.9	101	122.4	121.5	107.1
SEM+	1.5		1.0	1.2	1.4
n++	8		19	17	14
Sodium	138.6	138	152.0	150.3	15.8
SEM	1.9		1.8	2.1	1.6
n	14		17	18	12
Potassium			4.2	4.8	144.4
SEM			0.5	0.4	4.0
n			11	13	10
K+Na—Cl		41**	34	34	53

\+ Standard error of the mean.

++ Number of animals.

* Because the average serum protein was only 4.3 per cent in these young guinea pigs, calculation was based on a Donnan ratio of 0.96 and a water content of 965 gm. per liter.

** Assuming 4 m.Eq. K per liter of serum.

TABLE III.

INDIVIDUAL ELECTROLYTE VALUES IN SERUM, SPINAL FLUID, PERILYMPH AND UTRICULAR ENDOLYMPH FROM FIVE GUINEA PIGS.

All values expressed as milliequivalents per liter.

Guinea Pig No.	Chloreid				Sodium				Potassium		
	Serum	Sp. Fl.	Peri	Endo	Serum	Sp. Fl.	Peri	Endo	Sp. Fl.	Peri	Endo
48	91	123	120	107	134	156	160	5.6	6.8	6.3	170
49		118	121	106		146	150	19.1	6.7	7.3	143
54		121	122	116		148	144	16.2	3.9	4.4	146
57		121	124	112	149	164	160	16.6		6.0	141
59	94	122	125	111	130	152	154	12.7	2.2	2.7	143

per cent lower in endolymph than in perilymph. No difference was found in the concentration of any of the three ions between perilymph and spinal fluid. It will be noted that the concentration of undetermined anion is substantially greater in endolymph than in the other fluids.

The analyses of *cochlear endolymph* (Table IV) are fewer in number, and the collection of uncontaminated endolymph was much less successful than in the case of the utricle; nevertheless, there are six analyses for Na or K which concur with the utricular data. In an additional case (#101) the

data are much as would be expected from a mixture of endolymph and perilymph. Thus it seems quite probable that cochlear endolymph shares with utricular endolymph the unusual Na and K composition.

TABLE IV.

ANALYSIS OF COCHLEAR ENDOLYMPH.

All values are expressed as m.Eq. per liter.

Probably endolymph*			Possibly a mixture of endolymph and perilymph**			Composition not determined***		
Animal	K	Na	Animal	K	Na	Animal	K	Na
No. 101	49	116	No. 104		12	No. 89	109	
No. 106		38	No. 105	10	153	No. 98	175	12
No. 111	150		No. 109	10		No. 107	13	148

* Reissner's membrane was intact. In case No. 101, two holes were drilled into the bony cochlea. The first hole, in the third turn, was too large and the strial exposure considered disadvantageous. Fluid was removed from a second hole in the second turn. Sections revealed disorganization of the stria vascularis in the third turn, with some of the cells disrupted and free in the scala. Leakage from the spiral ligament into the scala media is not excluded.

** Based on observations during the fluid removal or on examination of the sections. Reissner's membrane was intact.

*** Inadequate fixation, distortion, or loss of the celloidin sections.

Protein analyses of labyrinthine fluids are particularly liable to disturbance from contamination with traces of blood. The data (Table V), therefore, set an upper limit to the concentrations of protein present, and the lowest values are perhaps the most reliable. It would appear that all three fluids are very low in protein, that endolymph contains no more protein than spinal fluid, but that perilymph may contain twice as much protein as either of the other fluids.

TABLE V.

PROTEIN CONTENT OF SPINAL AND LABYRINTHINE FLUIDS.

All values expressed as mg. per cent.

Spinal fluid	75,* 24, 24, 13, 21, 28,* 25, 19.	Av.** 21 ± 2
Perilymph	32, 19, 55, 33, 49, 65, 71, 56, 65, 60.	Av. 50 ± 5
Endolymph (utricular)	16, 97,* 11, 14, 26,* 72,* 34,* 152,* 18.	Av. 15 ± 2

* Indicates presence of microscopically visible red blood cells.

** Averages of those not contaminated with red blood cells. The standard error of the mean is indicated.

DISCUSSION.

The present study seems to demonstrate the advantage of the utricle as a source of endolymph, uncontaminated by perilymph. The utricle represents an easily accessible cistern of endolymph. The entire structure can be exposed so that the tip of the pipette is visible at all times. The surface of the

utricle can be kept free of fluid for an adequate period of time.

Several of the animals seem to present evidence for the functional importance of the utriculo-endolymphatic valve. This valve, which is an epithelial fold interposed between the utricle and the utricular duct was described by Bast.[18] Observations as to its functional capacity in retaining normal pressure in the utricle and semicircular canals have been made by Bast,[19] Bast and Eyster,[20] and Perlman and Lindsay,[21] The sections from three animals showed that the vestibular cochlear duct had been opened during the surgery; yet, the utricles showed normal distension. Judging from the chemical analyses, no mixing of perilymph and endolymph had occurred. These findings appear to present additional evidence for the ability of the utriculo-endolymphatic valve to close off the utricle and semicircular canals from the rest of the membranous labyrinth when normal pressure relations are changed.

The chemical findings agree with those of Kaieda,[1] for the shark, in regard to the similarity of spinal fluid and perilymph, but disagree in regard to the composition of endolymph (Table I). It is possible that the labyrinthine fluids of the fish might represent an adaptation to environmental and anatomical differences. The somewhat lower chloride found in endolymph is not in agreement with the data of Ledoux[8] for the cat (Table I).

The data presented show that endolymph, at least in the guinea pig, is unique among known extracellular fluids and resembles intracellular fluids in regard to Na and K content. The significance of this unusual electrolyte pattern is not obvious. It is difficult to reconcile the high K concentration of endolymph with the positive DC potential of the scala media demonstrated by Bekesey.[22] The situation is just the reverse of that found for muscle and nerve, in which a negative potential is associated with the side of the membrane having a high K concentration. Any attempt to resolve this difficulty must take into consideration the structure of the scala media. The stria vascularis at its lateral wall and the complex sen-

sory structure inferiorly may well add to the complexity of the processes occurring inside. It is possible that the DC potential and the K/Na ratio are related in some other way than in nerve and muscle fiber.

If the prevalent concept of the basilar membrane as one boundary of the cochlear duct is accepted and the tunnel is supposed to be filled with endolymph, then it would seem that the high K content should seriously interfere with the conduction of the nerve fibers that cross the tunnel. Davis, Tasaki and Eldredge[23] believe that their experiments with the injection of KCl into the scalae and their measurements with an exploring electrode show that the tympanic boundary of the cochlear duct should be placed at the reticular lamina covering the organ of Corti. The nerve, therefore, would be separated from the endolymph. The fact that the nerves do conduct impulses in spite of the high K content of endolymph clearly supports this concept.

SUMMARY.

Perilymph, cerebrospinal fluid, and endolymph from the utricle of the guinea pig were analyzed for Na, K, Cl, and protein. Cochlear endolymph was analyzed for Na and K, and blood serum was analyzed for Na and Cl. Special microchemical methods were devised for the purposes, since it was necessary to work with samples of the order of 0.2 μl. (.0002 ml.). Endolymph was found to have a K concentration 30 times that of perilymph, and a Na concentration only a tenth as high as perilymph or cerebrospinal fluid. The Cl of endolymph was found to be 90 per cent of that in perilymph and cerebrospinal fluid. Perilymph and cerebrospinal fluid were indistinguishable in K, Na, and Cl content. All three fluids are low in protein, but perilymph may contain twice as much as either of the other fluids.

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LARYNGEAL SPASM.*†

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INTRODUCTION AND DISCUSSION.

This is a report of work undertaken to secure, if possible, direct evidence on the nature of laryngeal spasm. We proceeded on the basis that laryngeal spasm depends upon the operation of the usual mechanism for protection of the trachea and lungs by glottic closure. This involves an understanding of the anatomy and physiology of the larynx. The following scheme was used as a guide:

I. Structures involved: A. Nervous—

1. Afferent pathways—
   a. From the surface of the pharynx and the larynx *i.e.* Cranial nerves, V, IX, and X.
   b. From any other structure (in theory at least any afferent nerve may be involved).
2. Central apparatus (*i.e.* the entire central nervous system) connecting the pathways noted above with the
3. *Efferent* pathways in cranial nerve X (vagus).
   a. The superior laryngeal nerve (external branch to the cricothyroid muscle).
   b. The recurrent laryngeal nerve (branches to all other laryngeal muscles).

B. Muscular—

The various muscles, abductor and adductor of the glottis. These are *striated* (skeletal or voluntary) muscles.

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Although the Xth cranial nerve (vagus) is the important connector structure, it is well to note that neither the innervation nor the effectors (muscles) are autonomic.

The peripheral nerves are *medullated;* there are no *peripheral efferent ganglia;* and the muscles are *striated.*[3,4,5] There is, therefore, no reason to expect that autonomic blocking agents (*e.g.* atrophine) will have any direct effect on the structures involved.

II. Physiology—A discussion of the innervation of the larynx with bibliographies is given by Cole[1] and by Murtagh.[2] The largest *adductor* of the glottis is the cricothyroid muscle which is supplied by the external branch of the superior laryngeal nerve. The important *abductor* is the posterior cricoarytenoid muscle supplied by the recurrent nerve.

In brief, laryngeal spasm may be orginated

1. *Reflexly*—
   a. By intensive stimulation of pharyngeal or laryngeal receptors
   b. By intense (painful?) stimulation of any receptor, (6) (7);
2. *Centrally* by altering the state of the central apparatus (*e.g.* strychnine poisoning); and
3. *Peripherally*—
   a. By stimulating an efferent nerve, or
   b. By stimulating the muscles directly.

Laryngeal spasm may be prevented or stopped by avoiding the circumstances listed above. This may be accomplished in

1a. By local anesthesia of the pharynx and the glottis or more immediately by not allowing secretions to accumulate; *1b.* and *2.* By adequate general anesthesia; and *3.* By avoiding trauma in the neck region.

The effects of *1, 2,* and *3a* may, of course, be prevented by the use of a neuromyal blocking agent (*e.g.* curare).

Proper use of an intratrachial tube obviously avoids most difficulties.

METHOD.

Goats were used as experimental animals. The basic anesthesia was pentobarbital sodium (Nembutal) given intraperitoneally (0.45 Gm/kilo).

A tracheal cannula was inserted well down in the neck to leave the upper trachea and larynx free for experimental procedures. The recurrent nerve was visualized on each side, and the esophagus was tied to prevent regurgitation. The pharynx, in most experiments was packed with gauze in an attempt to keep secretions from entering the glottis.

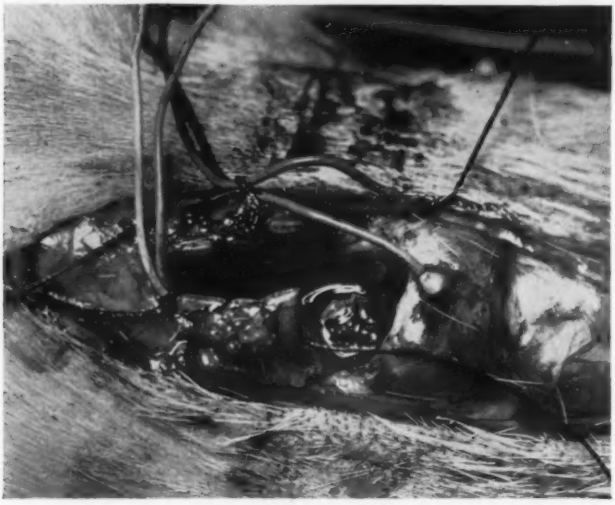

Fig. 1. A Photograph showing the Placement of the Needle Electrodes.

The trachea was opened between the second and third rings, taking care to avoid the recurrent nerve. The larynx was elevated and the dorsal portion of the cricoid cartilage freed to expose the posterior cricoarytenoid muscles bilaterally.

Electrodes were inserted in each *posticus* muscle (an abductor); the larynx was laid back in place; and the lead wires from the electrodes were anchored by a suture around the third tracheal ring. Similar electrodes are placed in each cricothyroid muscle (an adductor) (see Fig. 1).

The electrodes are made by soldering insulated, flexible lead wires to the hubs of 31-gauge intradermal needles. The needles (hubs included) are then dipped in insulating varnish (Tygon). When the varnish has hardened a length of 1 to 1.5 mm. at the point is bared by dipping in solvent. The needles are inspected under a microscope and are tested by passing direct current electricity at 500 volts between the needle and a strip of metal (anode), with both immersed in a dilute saline solution. If bubbles form on the needles anywhere except on the bared tip the electrode is recoated.

A large indifferent electrode is fastened to shaved skin between the rami of the lower jaw, using collodion. A ground electrode is placed in a similar manner on the vertex.

The action potentials of the individual muscles are recorded against the potential of the indifferent electrode by leading to an eight channel Grass Model III encephalograph. Any potential change which is recorded by all channels simultaneously will have its origin under or near the indifferent electrode, and will probably represent a swallowing motion.

The placement of the individual electrodes is facilitated by temporarily connecting each electrode to a separate preamplifier, which is in turn connected with both a cathode ray oscilloscope and an audio amplifier—loud speaker set-up. While viewing the pattern on the scope and listening to the sounds from the speaker, the electrode is manipulated until it is near an active motor unit.

The recorded electrical response is principally from the units in the neighborhood of the electrode. Repeated experiments, however, should give a reasonable picture of the response of the muscle as a whole.

An accordion type pneumograph is placed around the chest, connected with a strain gauge bridge, so arranged that the circumference of the chest is represented by an envelope described by one of the pens of the encephalograph.

A microphone connected with one channel of the encephalograph is placed over the tracheal cannula to indicate the instant and, in a general way, the force of the expiratory blast.

EXPERIMENTAL RESULTS.

Controls and Local Reflexes—

Until we had considerable experience with the preparation; had learned to keep the glottis scrupulously clean, and had arranged to deliver the pentobarbital sodium continuously through an intravenous cannula, we secured no records from the muscles which were not indicative of continuing contraction (spasm). Records obtained from a preparation showing

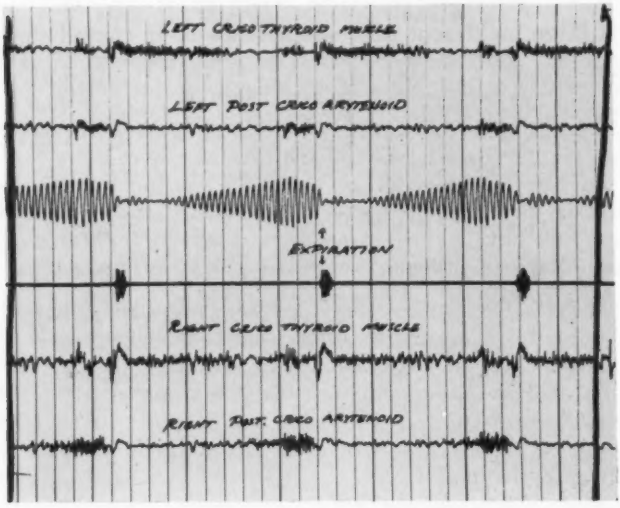

Fig. 2. Experiment of July 9, 1953 — Goat. Channels 1, 2, 5 and 6 Electromyograms, Channel 3 envelope of chest size, Channel 4 microphone showing Expiratory blast. The time interval between vertical lines is 0.4 sec.

what we consider to be a normal respiratory pattern are shown in Fig. 2. Here the muscle action potentials are shown during quiet respiration. Throughout expiration the cricothyroids (adductors) show moderate activity. The greatest activity of the posticus muscles (abductors) is late in inspiration. Synchronous with the activity of the posticus group there is also some slight discharge from the cricothyroids. Both muscle groups show periods of relative inactivity.

The electrical activity, when spasm is present, is shown

in Fig. 3. Here there is no evidence of respiratory rhythm in the action potential records. The potentials are increased, and the firing continues undiminished throughout the respiratory cycle. This record was taken just before alcohol was applied to the right recurrent nerve. Fig. 4 shows the cessation of activity in the right posticus five minutes after the

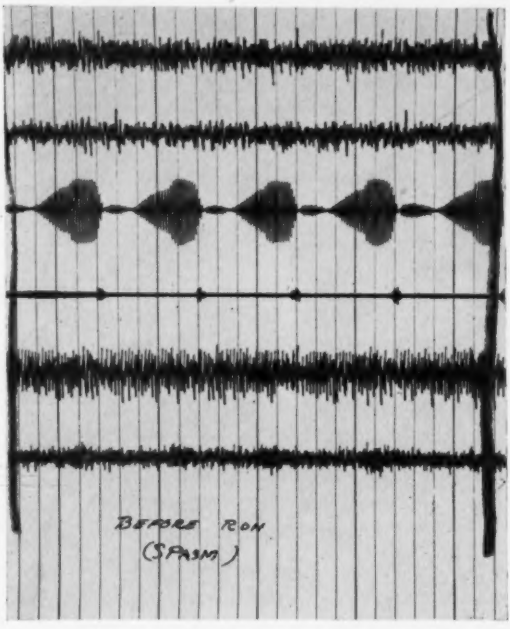

Fig. 3. Experiment of July 9, 1953 — Goat. Channels as in Fig. 1. Time interval between vertical lines is 1.0 sec.

application of alcohol to the nerve. As a further control the left recurrent nerve was cut. The results are shown in Fig. 5. There are no action potentials from the right posticus muscle (previous alcohol block), and the action potentials from the left posticus cease on section of the nerve. There is a flurry of activity in the left cricothyroid at the moment of section, followed by reduced activity of both cricothyroids for a period of 0.10-0.12 seconds. We are at present uncertain of the explanation of this transient inhibition.

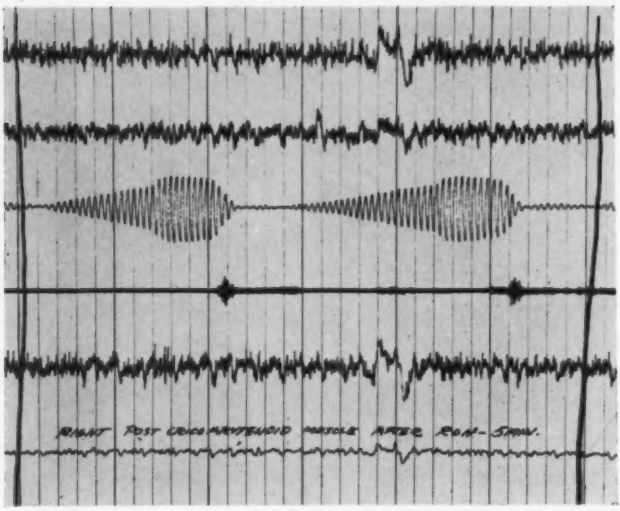

Fig. 4. Experiment of July 9, 1953 — Goat. Channels as in Figs. 2 and 3. Time 0.4 sec. between lines.

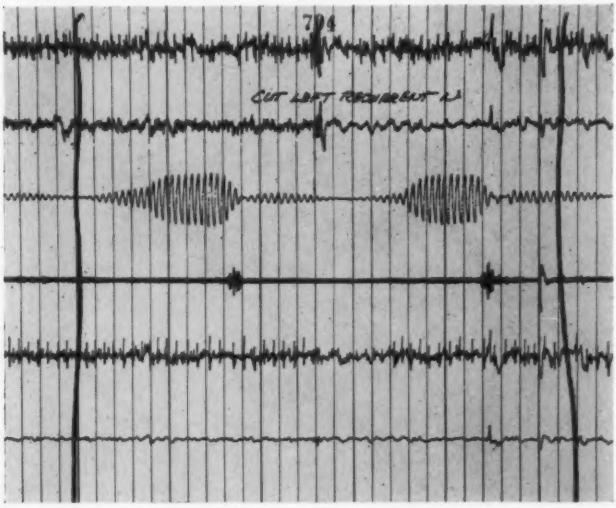

Fig. 5. Experiment of July 9, 1953 — Goat. Details as in Fig. 4.

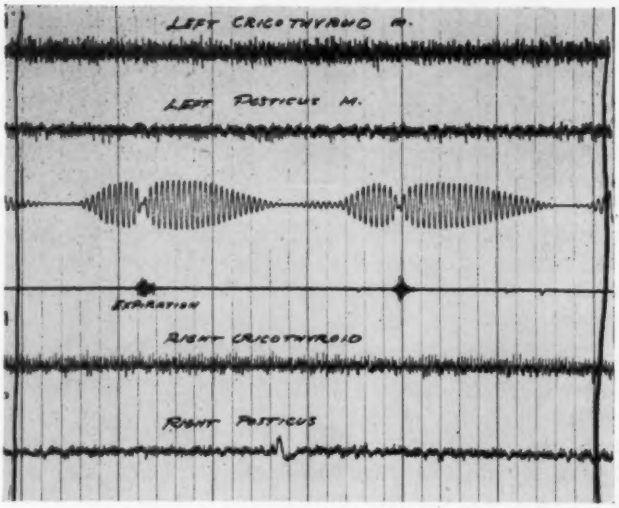

Fig. 6. Experiment of July 6, 1953 — Goat. Channels as in Fig. 2. Time 0.4 sec. between lines.

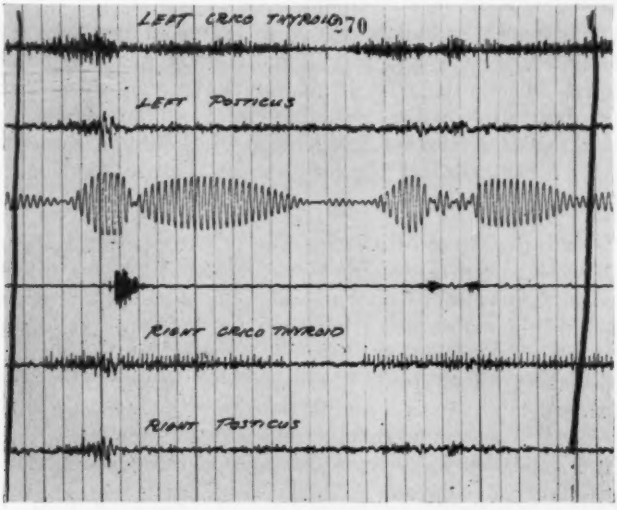

Fig. 7. Experiment of July 6, 1953 — Goat. Channels and Time as in Fig. 6.

The effect of foreign material in the glottis is shown in Fig. 6. The electrical records indicate continuing contraction of all four muscles. The respiratory record is marred by the fact that the pneumograph apparatus is not in balance, and that both inspiratory and expiratory movements appear as apparent increases in circumference of the chest. The microphone record, however, gives a correct indication. After the larynx had been freed of blood clots and saliva, washed with saline and sucked dry, the character of the responses changed to the pattern shown in Fig. 7. Here rhythmic respiratory changes in action potentials replace the spasm shown in the previous figure.

The effects of cutting the internal (sensory) division of the superior laryngeal nerve is shown in Fig. 8. Here there is a transient inhibition of the activity of the muscles, particularly of the cricothyroids. Recovery is nearly complete at the end of a minute (see Fig. 9). Twenty seconds later spasm had returned and the record resembled Fig. 8.

EFFECTS OF ANESTHETIC AGENTS.

Pentobarbital Sodium (Nembutal)—

Pentobarbital sodium was used as the usual agent, both for induction and maintenance. With a good preparation and adequate anesthesia, records similar to those shown in Fig. 2 were obtained. To note the effects of other agents the continuous, dilute, intravenous pentobarbital was disconnected, and the other material injected through the polyethylene tube in the femoral vein.

To note the effect of a large dose of pentobarbital, the animal was allowed to become light and the drug was injected between the arrows as shown in Fig. 10. Because of improper switching the action potentials are recorded from the left and right cricothyroid muscles only. The remaining two channels are also left cricothyroid. Chest size and the expiratory blast are shown in channels 5 and 6, the slower swings (synchronous in all electromyograms) represent swallowing. Within eight seconds all activity in the muscle records ceased and ventilation had stopped. Artificial respiration was given for thirteen minutes, when spontaneous respiration again began.

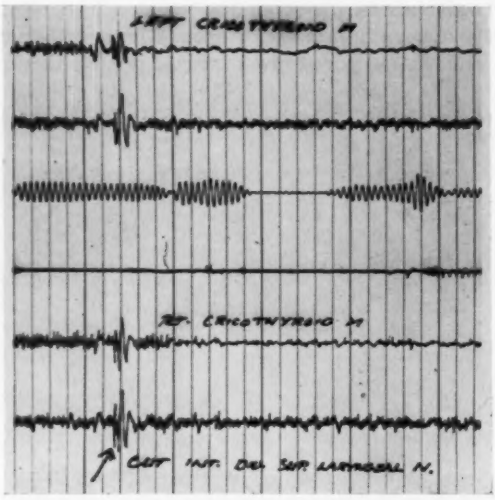

Fig. 8. Experiment of July 6, 1953 — Goat. Channels as in previous figures. Time 0.4 sec. between vertical lines.

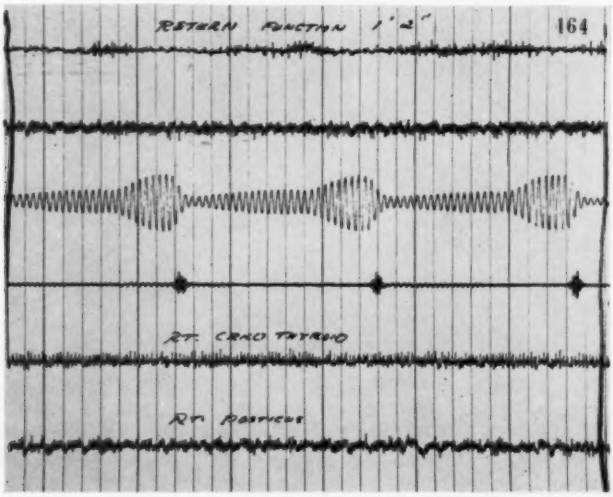

Fig. 9. Experiment of July 6, 1953 — Goat. Channels and Time as in Fig. 8.

During this time, as observed on the oscilloscope and loud speaker there were no action potentials from either cricothyroid, and only a few small disturbances from either posticus

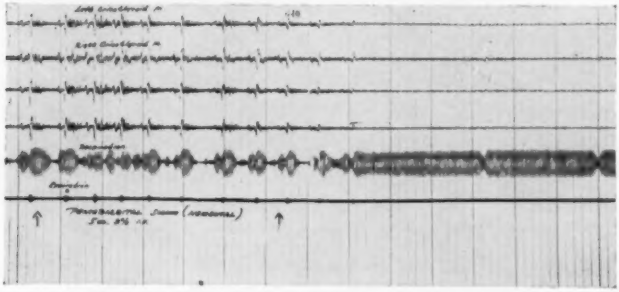

Fig. 10. Experiment of December 3, 1953 — Goat. Time 1 sec. between vertical lines. See text for description.

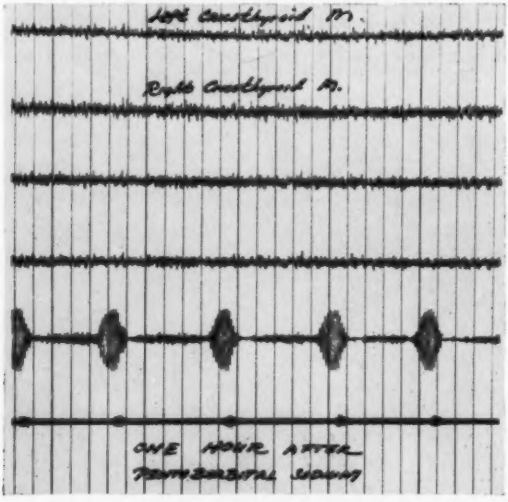

Fig. 11. Experiment of December 3, 1953 — Goat. Details as in Fig. 10.

At the end of eighteen minutes, although respiration was continuing, there were no evidences of activity of the glottic musculature. One-half hour after the injection there were a few rhythmic bursts of muscle potentials, and after an additional

five minutes (no anesthesia during this time) continuous firing (spasm) was resumed. Figure 11 shows conditions at the end of an hour. After some thirty-five minutes the slow intravenous pentobarbital had been resumed. These results are typical of our experience with pentobarbital.

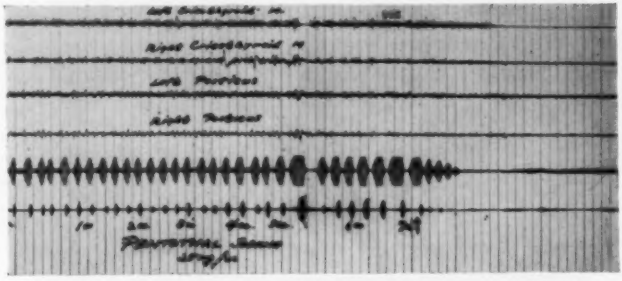

Fig. 12. Experiment of December 10, 1953 — Goat. Myograms as labelled with Respiration and Expiratory Blast in Channels 5 and 6. Rate of injection shown on record.

Pentothal Sodium—

This agent is reputed to cause laryngospasm in some experimental animals[7] and has a bad name in this respect among anesthesiologists. We made some half dozen experiments, using it, and will report on one run only. In Fig. 12 is a record of an injection, and some twenty seconds after the administration. The animal, deliberately light, showed electrical evidences of continuing activity, upon which was superimposed a respiratory rhythm. The record is sufficiently graphic and shows that ventilation stopped within five seconds of the final cc. of pentothal, and that all large potentials had disappeared within ten seconds. Inspection showed the cords motionless. Artificial respiration was carried on for about four minutes. Respiratory rhythms began in the myograms at that time, and ventilation resumed in about a minute. The conditions at the end of eight minutes showing a slight reduction in electrical activity, particularly in the abductors, is shown in Fig. 13. In none of the experiments was there any evidence of increased electrical activity of the muscles.

No other anesthetic agents were tried.

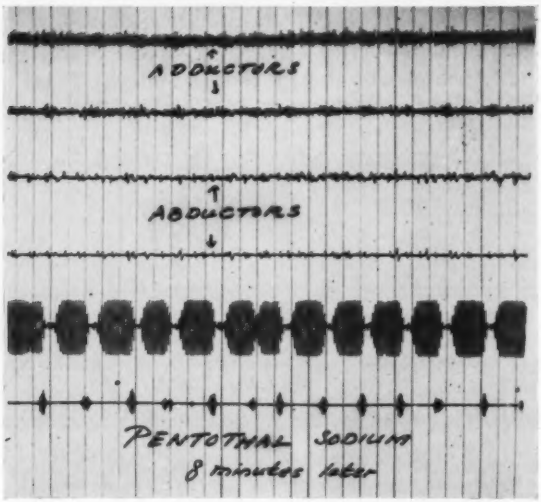

Fig. 13. Experiment of December 10, 1953 — Goat. Channels as in Fig. 12. Time 0.4 sec. between lines.

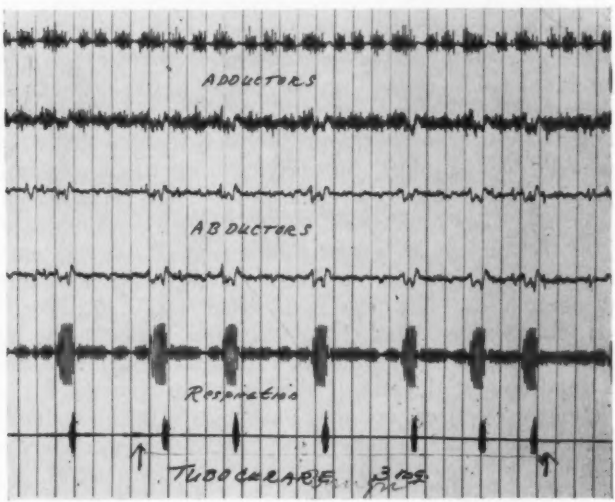

Fig. 14. Experiment of December 10, 1953 — Goat. Details as in Fig. 13.

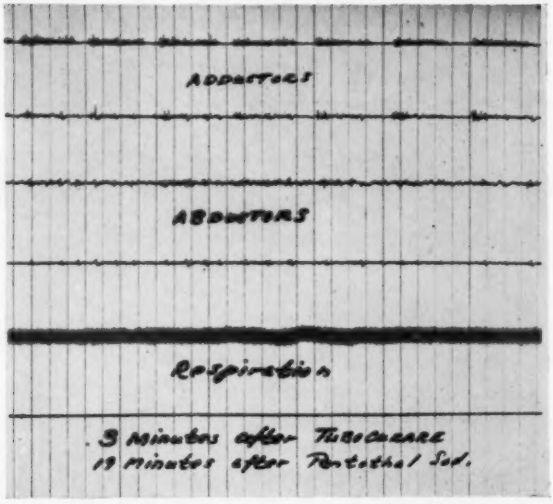

Fig. 15. Experiment of December 10, 1953 — Goat. Details as in Fig. 13.

MYONEURAL BLOCKING AGENTS.

Curare—

In several experiments curare (tubocurarine chloride) was injected in various amounts and at various rates. In every case respirations stopped, and the action potentials from the glottic muscles diminished, and then disappeared. The records of Figs. 14 and 15 were chosen because they represent a continuation of the records shown in Figs. 12 and 13. The curare was given sixteen minutes after the pentothal. In just over one minute the respirations had stopped, and the muscle potentials were reduced, although they still showed respiratory rhythms. At the end of three minutes the changes are shown (see Fig. 15). Artificial respiration was not given. The electromyograms vanished in six minutes, although swallowing potentials continued for several more minutes.

Succinylcholine—

Succinylcholine chloride (Anectine) was given on several occasions. Although its mode of action is considered to be quite different from that of curare, the results are very sim-

ilar. Respiration was depressed before any changes were noted in the electrical records from the glottis. In some experiments, particularly when using larger doses, there was evidence of a transient augmentation of potentials (in one case a slight, general convulsion) before the depression set

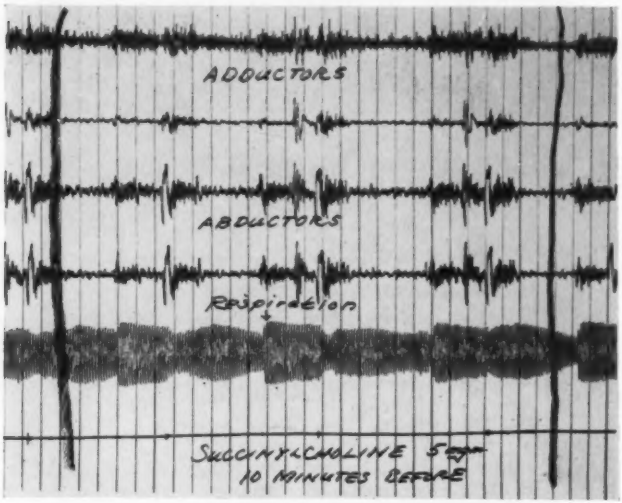

Fig. 16. Experiment of December 17, 1953 — Goat. Details as in Fig. 13.

in. From the records on hand, one set is chosen because it illustrates a number of points. A moderate dose (5 mgms. diluted 1 to 5) was slowly injected but not washed in. Within one minute respiration had stopped, though the action potentials of the glottic muscles continued. Artificial respiration was given for four minutes when shallow ventilation returned. Evidences of increased glottic activity appeared, and the condition at the end of ten minutes is shown in Fig. 16. At this time the cords were abducted. The intravenous pentobarbital, which had been stopped during this time, was then started. The succinylcholine remaining in the tubing was in this way washed into the circulation.

We have no way of knowing how the dose was divided. The electrical changes in the glottis continued as respiration was

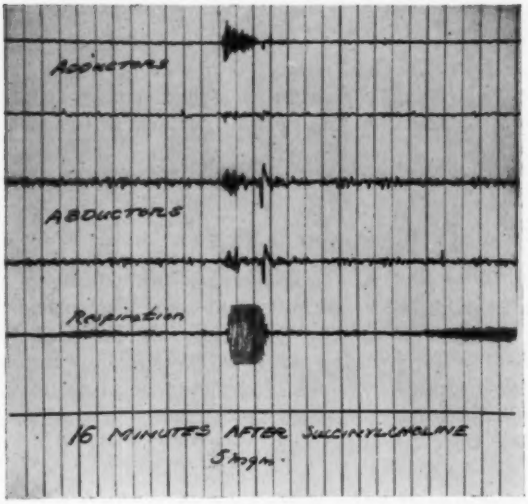

Fig. 17. As in Fig. 16.

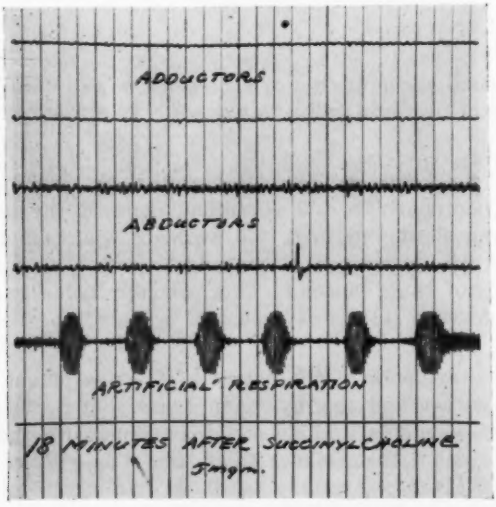

Fig. 18. As in Fig. 16.

again depressed. Six minutes later the record of Fig. 17 was obtained. Respiration is very slow, the expiratory blast is negligible, but the action potentials are certainly not diminished. Figure 18 shows the record at the end of eighteen minutes. Artificial respiration was required for an additional five minutes, and then the normal pattern returned.

No other blocking agents were tried.

CONCLUSIONS.

Using the electrical changes which take place in striated muscles as evidence of activity, and considering that the size and the frequency of these potentials are indications of the quantity of activity taking place, we have studied the behavior of the glottis under various circumstances. Because of technical difficulties only two pairs of muscles (one adductor and abductor) were investigated.

With the data at hand it has been shown that experimental laryngospasm may be produced reflexly by stimulation of the laryngeal mucosa (or any sensory nerve leading from the larynx).

The usual accumulation of secretions is adequate to set up this spasm, if the depth of anesthesia is inadequate.

No evidence was obtained to show that the fixed dose anesthetics, pentobarbital and pentothal, increase the activity of the laryngeal musculature. All evidence was that these materials given in adequate amounts depress (centrally) the responses of these muscles. This probably holds true for all central nervous system depressants.

Graphic evidence of the action of the neuromyal blocking agents (curare and succinylcholine) has shown the effectiveness of these agents, and has strikingly illustrated the respiratory depression that follows their use. The glottic musculature fails after respiratory paralysis has set in and before swallowing movements have stopped.

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THE INCREASING INDICATIONS FOR TRACHEOTOMY.*†

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In 1940, one of us (G.S.F.) presented a study of 100 patients[1] upon whom 102 tracheotomies had been performed. The primary motive for this survey was to learn something of the risks and complications resulting from such a procedure. The results indicated that the risks involved in this surgical procedure were negligible, and that the only complication occurring with any degree of frequency was pneumothorax; however, the pathologic condition for which tracheotomy was necessary was responsible for a rather high incidence of death in this group.

Again after a period of some fourteen years the same type of study has been made, with some difference in that the primary aim in this later group was to examine the indications for tracheotomy. It is apparent to all practicing medicine and surgery now, that these indications have increased in scope and number in the last few years. Perhaps it is better stated, that these reasons have always existed, but that the recognition of such recently, has come with a better understanding of the physiology of respiration as applicable to clinical, rather than laboratory medicine. The main impetus for the development of this additional knowledge results from the opportunity to study the respiratory problem afforded by the many unfortunate poliomyelitis victims. Credit must be given to Wilson,[2] Galloway,[3] Cummings,[4] Priest[5] and Bower,[6] their co-workers and others, for their invaluable contributions to this subject. Harris[7] in 1952 published the

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results of his efforts to investigate the clinical value of tracheotomy in the prevention and management of the various types of pulmonary difficulties encountered in poliomyelitis. His conclusions were that a reduction in poliomyelitis fatalities could be accomplished by tracheotomy in certain cases, particularly when performed early. This has been the experience of many other otolaryngologists.

The need for tracheotomy for obstructive conditions in the hypopharynx and larynx, due to edema of the mucous membranes, as the result of inflammation, allergies, trauma and chemicals, is well established and recognized. Intrinsic mechanical obstruction, due to vocal cord paralysis, foreign bodies and tumors is commonplace; extrinsic pressure from cervical masses, neoplastic or inflammatory in nature, causing embarrassment in respiration, is not unusual.

As mentioned, there has been an increase in the indications for tracheotomy in recent years. We have noticed this in our experience, as will be presented later, and we have also observed this trend recorded in the literature.[8,9] Specifically, Carter and Giuseffi[10] have reported the use of tracheotomy in chest injuries; Atkins,[11] in the prevention of pulmonary complications in postoperative and debilitated patients; Lahey and Hoover,[12] following thyroidectomy; Echols[13] and McCart[14] and their co-workers in the management of head injuries; Herzon,[15] Creech[16] and their collaborators in the treatment of tetanus; Taylor and Austin[17] in the handling of pulmonary complications in neurosurgical patients; Collins, et al.,[18] in eclampsia; Sloan,[19] in leprosy; Bofinkamp,[20] in botulism; Lewy[21] in barbiturate poisoning; Everett[22] in laryngotracheobronchitis, and Colvin and Morrison[23] in acutely ill patients.

The basic reason for tracheotomy is to permit a normal exchange of air in the alveoli of the lung for the absorption of oxygen and the elimination of carbon dioxide (Harris[7]). Any significant interference with this mechanism will result in rapid death, or if corrected just prior to this stage, very possibly in irreversible nerve tissue injury, with death secondary to complications therefrom. If the patient survives, permanent embarrassment and compromise in the efficient

function of the human mechanism may ensue. Anoxia, hypoxia, carbon dioxide retention in the blood and acidosis are not tolerated for long periods. In this regard, it has become apparent that obstructive conditions, such as retained tracheobronchial secretions, may well lead to subclinical anoxia and hypercapnia with cerebral manifestation, which in turn may be confused with, or be blamed upon, the primary agent, such as infection (encephalitis) or hemorrhage (trauma). The correction of the responsible medium producing the anoxia by establishment of an adequate airway may cause a marked difference in the future well-being of the patient.

A total of 150 consecutive patients upon whom tracheotomies had been performed, were studied. Two patients in the group required two procedures; the second one in each case was performed prior to laryngeal surgery, which had been unsuccessful on the first occasion. Thus a total of 152 tracheotomies were available for consideration; however, the two extra procedures were not considered specifically in our statistics, as such detail seemed irrelevant. The statistics upon 150 tracheotomies are presented.

In our survey we have divided the indications for tracheotomy into three known groups, with a possible fourth group as a questionable and poorly understood one (see Table I):

TABLE I.

INDICATIONS FOR TRACHEOTOMY.

1. Fixed Obstruction to Upper Airway. (Rapid Obvious Anoxia)		
2. Fluid Obstruction to Lower Airway. (Slow Obscure Anoxia)		
3. Prophylactic. (To prevent 1 and/or 2)		
Total Number of Patients Having Tracheotomies		150
1.	80	53.0%
2.	30	20.0%
3.	40	27.0%

First were those that exhibited the manifestations of an impaired airway with anoxia due to more or less fixed mechanical obstructions, such as encountered in a neoplasm of the larynx or bilateral cord paralysis. The degree of impairment varied, of course, but in all cases tracheotomy was indicated. There is little room for confusion in this group.

The *second group* included those cases in which the airway was compromised by a fluid mechanical obstruction, such as an excessive accumulation of material in the tracheobronchial tree from repeated aspiration of oral fluids, hyperfunction of the secretory elements of the tracheobronchial membranes, and/or failure in the cough mechanism.

The *third group* was composed of those cases in which it was anticipated that the airway would eventually be inadequate because of the development of one or both of the conditions exemplified in the first two groups, as for example, in surgery of the upper respiratory or digestive tract such as an arytenoidectomy or extensive cervical dissections. Tracheotomy was performed as a prophylatic measure.

Spasm of the larynx superimposed upon some of the conditions previously mentioned and augmenting respiratory distress undoubtedly hastens the necessity for tracheotomy. We doubt seriously that spasm of the larynx *per se* will cause sufficient trouble to be classified as an indication for tracheotomy, although we certainly do not feel secure in this opinion at the present time.

As one would anticipate, in a general hospital with an active otolaryngologic service, the larger group of tracheotomies was in the static obstructive, or first category (see Table II). Eighty patients were so classified. The obstruction in 23 patients was due to carcinoma of the larynx, or of some structure above the larynx. In 22 incidents, the obstruction was the result of inflammatory conditions such as laryngotracheitis, diphtheria, Ludwig's angina, and one unusual case of pemphigus. As the result of surgery upon the thyroid gland (non-malignant), 11 patients required tracheotomy because of subsequent obstruction to the airway.

Nine children who were admitted with pulmonary foreign bodies, needed additional airway afforded by tracheotomy because of the nature of, and the reaction to, the foreign

TABLE II.
INDICATIONS FOR TRACHEOTOMY.

1. Fixed Obstruction to Upper Airway. Conditions:	Number of Patients
Carcinoma	23
Inflammatory	22
Thyroid Gland Surgery	11
Pulmonary Foreign Bodies	9
Trauma	4
Bilateral Cord Paralysis	4
Papillomata, Larynx	2
Miscellaneous:	
Stenosis (scar), Larynx	1
Gumma, Larynx	1
Ulceration (Chemical), Larynx	1
Pyogen. Granuloma, Larynx	1
Edema (Endotr. Tube), Larynx	1
	80

body, plus the edema incurred as the result of instrumentation in its removal. Trauma to the airway from external forces, such as sustained in automobile accidents, was responsible for the procedure in four incidents. Tracheotomies were performed upon four patients with bilateral vocal cord paralysis, the causes of which were obscure. Two children with papillomata of the larynx needed the augmented airway afforded by tracheotomy.

Single cases requiring tracheotomy were: stenosis of the larynx, due to scarring as the result of X-ray irradiation and chondritis, gumma of the larynx, pyogenic granuloma of the larynx, ulceration secondary to the ingestion of bichloride of mercury, and laryngeal edema secondary to prolonged endotracheal intubation used as an adjunct in the administration of an anesthesia.

In the second group of cases (see Table III), tracheotomies were performed in order to facilitate the cleansing of the fluid collection in the tracheobronchial tree and thus prevent anoxia from this source. Thirty patients were placed in this category, somewhat less than had been anticipated. Neurological conditions, such as spinal cord and brain le-

sions, were responsible in seven cases. Trauma to the lower face, neck and chest necessitated tracheotomies in eight patients. A medical group composed of diseases such as polio-

TABLE III.
INDICATIONS FOR TRACHEOTOMY.

2. Fluid Obstruction to Lower Airway. Conditions:	Number of Patients	
Neurological Group		
Brain Tumors	3	
Amyotrophic lat. Sclerosis	1	
Postop. Lobotomy	1	
Guillain-Barre Synd.	1	
Cardiac Arrest.	1	7
Medical Group		
Poliomyelitis	6	
Uremia	3	
Meningitis	2	
Tetanus	1	
Pneumonia	1	13
Traumatic Group		
Face, Neck, Chest	8	8
Miscellaneous Group		
Neoplasm of Mouth	1	
Amyloidosis, Trachea	1	2
TOTAL		30

myelitis, meningitis, pneumonia, uremia, and tetanus were responsible for 13 procedures. The remaining two cases were: one of amyloidosis of the trachea and one of oral neoplasm.

TABLE IV.
INDICATIONS FOR TRACHEOTOMY.

3. Prophylactic Conditions:	Number of Patients
Extensive Head and Neck Surgery	35
Traumatic Surgery	4
To Facilitate Anesthesia	1
TOTAL	40

There were 40 cases in the third classification (see Table IV). It is in this group that tracheotomy was performed prior to the development of conditions for which tracheotomy was performed in Groups I and II. In other words, this was the group in which prophylactic tracheotomy was in order. Practically all of the patients in this category were those

requiring extensive surgery upon the upper respiratory and digestive tracts, such as the tongue, jaws, etc., in an effort to eradicate carcinoma. In a few this was performed at the time of repairing injuries secondary to trauma. One particular case in this class is worth mentioning, in that in our experience it was the first time such an indication had occurred: An underdeveloped and ill 9-months-old child was tracheotomized prior to chest surgery in order to facilitate the administration of a general anesthetic.

From the above-mentioned cases, we see a more liberal use of tracheotomy with an increase in the number of indications, as exemplified in Groups II and III. We are not certain but that these conditions have been considered as indications all along, to a certain degree, and that the increase is relative, in that the added publicity has focused our attention upon this method of treatment. For instance, in our previous study, a case of poliomyelitis in 1933 received a tracheotomy in order to facilitate the cleansing of the tracheobronchial tree.

With the increase of poliomyelitis, there has been an increase in the number of tracheotomies in the treatment of this disease. Also, since 1933, there has been an actual decrease in conditions causing upper respiratory tract obstruction such as diphtheria, laryngotracheitis and foreign bodies. With the development of improved surgical techniques and antibiotics, more extensive surgical procedures are being carried out, and this is reflected in the enlarged number of prophylactic tracheotomies (third group).

In this recent study special care was made in an attempt to analyze the risk and complications of tracheotomies. This was done because some physicians consider the procedure a hazardous one. Apparently this is not true, for in not one case could we find that the procedure had prolonged the illness of the patient or had hastened death. Obviously, in the vast majority of cases, the opposite occurred. The only complications of note, occurring in this series as the result of tracheotomy *per se*, were mediastinal emphysema and pneumothorax.

Pneumothorax, recognized by radiological and clinical examination, was present in only three recorded cases. We believe that this figure is incorrect, because if routine chest X-ray examinations had been made following tracheotomy, it is likely that more cases of pneumothorax would have been disclosed. The three cases mentioned as recorded complications recovered promptly. Subcutaneous and mediastinal emphysema was often present, but not to a degree to cause concern.

It was recorded in one case that expired within 12 hours after tracheotomy, that more than the usual amount of blood was noted from the wound postoperatively, but that after careful consideration, it was the opinion of the attending staff that this excess blood loss was not a factor in the cause of death. The expected slight wound infection and tracheobronchitis, secondary to the mechanical trauma resulting from the cannula and aspirating tubes, were not considered as complications, as they never reached any degree significant enough to influence the clinical course of the patient.

There were no cases of mediastinitis, tracheal granuloma, or apnea following relief of the obstruction. Eight patients expired within a period of 12 hours after tracheotomy, death in each instance being due to the primary disease or condition requiring the operation. No difficulty was encountered in decannulating any patient in whom this was desired. Davis[24] and his group report in their recent study a higher percentage of complications, but theirs was made from a more critical viewpoint than ours.

As in our previous survey[1], an effort was made again to classify the cases according to the degree of respiratory difficulty requiring tracheotomy. We considered elective cases as those in which respiratory difficulty was not present, tracheotomy being performed in anticipation of such a status, or to facilitate future or present therapy. Emergency cases were those in which total asphyxia was imminent, and the procedure was carried out with little regard to sterile technique or to the assembly of any more of a set-up than that supplied by the emergency tracheotomy tray.

Between these two extremes was classified our so-called intermediate group, in which definite respiratory difficulty or anoxia was present, but to such a degree that the procedure could be planned and performed with the maximum degree of orderliness, safety, and comfort to the patient and surgeon. In the total survey of 150 cases, 72 were elective procedures, 51 were in the intermediate group, and 27 were classified as emergencies, four of the latter having been performed on the wards, or in the emergency room without benefit of the emergency tracheotomy trays.

Comment: A majority of the tracheotomies were carried out by otolaryngologists, but an increasing number were performed by the thoracic and general surgeons. Evidence was present that those performing the tracheotomies other than the otolaryngologists in some instances, were in need of more knowledge of the procedure to obtain the maximum benefit therefrom. This was particularly true in the after-care of the patient.

It is evident also that the indications for tracheotomy, as well as some basic principals of the technique and after-care, should be given more consideration in undergraduate teaching. Tracheotomy is occasionally the solution to one of the few true emergencies in the practice of medicine, respiratory distress, and every physician should be prepared to aid in the solution of the problem.

SUMMARY.

A survey was made of 150 consecutive patients who had undergone tracheotomy. In comparing this study with one made some years previously, it was evident that the indications for tracheotomy had increased, and it was also apparent that this increase was due, in the main, to a better understanding of the clinical physiology of respiration. The fact which had not been universally recognized previously, was that obstruction in the lower respiratory tract from a collection of fluid, could be responsible for anoxia and hypercapnia, as well as, but more insidiously than the well-recognized upper respiratory tract obstruction. Aspiration by way of tracheotomy is the most successful treatment of lower tract

obstruction, providing postural drainage, bronchoscopic aspiration, and aspiration by other methods have not proved effective or feasible.

An evaluation of the complications secondary to tracheotomy indicates that the procedure is not a hazardous one, but a relatively safe operation, offering many advantages to the patient.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

LOWER ESOPHAGUS PROBLEMS.*

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In considering lower esophagus problems this presentation is confined to a discussion of those symptoms caused by changes in the anatomy of the esophagus, diaphragm and cardia. Consideration of inflammatory lesions are limited to esophagitis seen in two cases, following left pneumonectomy and scleroderma of the lower esophagus, both suggesting a newer concept as to the etiology of esophagitis.

Bozer[1] in his article on "Endoscopic Problems" included an interesting excerpt from Thomas Willis'[2] book of 1679, describing the treatment for symptoms suggesting cardiospasm:

"No less well, a very rare case of a certain man of Oxford shew an almost perpetual vomiting to be stirred up by the shutting up of the left orifice. A strong man, and otherwise healthful enough, laboring for a long time with often vomiting, he was wont very often, though not always, presently to cast up whatsover he had eaten. At length the distress having overcome all remedies, he was brought into that condition, that growing hungry he would eat until the esophagus was filled up to his throat, in the meantime nothing sliding down into the ventricle, he cast up raw (or crude) whatsoever he hath taken in: when no medication could help and he languished away for hunger, and every day was in danger of death. I prepared an instrument for him like a rod of whale bone with a little round button of sponge fixed to the top of it; the sick man having taken down meat and drink into his throat, presently putting down in the esophagus he did thrust down into the ventricle, its orifice being open, the food which otherwise would have come back again; and by this means he hath taken of his sustenance for 15 years and doth yet use the same machine and is yet alive and well,

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who would otherwise perish for the want of food. Without doubt in this case the mouth of the stomach always closed, either by a tumor or palsie, nothing could be admitted into the ventricle unless it were violently opened." This problem is still with us some 274 years later.

Since we are considering problems caused by changes in the structures around the hiatus for the esophagus in the diaphragm, a brief review of the anatomy is in order. From extensive origins about the thoracic cage muscular fibres converge to a common insertion to form the central tendon. The two principal openings through the diaphragm, in which we are interested, are the hiati of the esophagus and aorta. Both are behind the central tendon, the esophagus being surrounded by a sphincter-like arrangement of the crural fibers and the aorta just posterior to the middle arcuate ligament. The esophagus and aorta are incorporated by a figure "8" musculofibrous investment. There is no demonstrable muscular connection of the esophagus and diaphragm, these being fused by considerable amount of strong connective tissue. The sphincter cardia is inferior to the diaphragm and below the cardiac antrum. This anatomical factor will be dealt with later in showing the various epi-diaphragmatic intrusions of the sliding hiatal hernia, hiatal hernia, and parahiatal hernia.

The mechanics of respiration are important by reason of the variations of intra-thoracic pressure. During a respiratory cycle the maximum change in pressure is from — 70 to + 100 mm. of Mercury. At the time of a severe cough this pressure excursion is sudden and not over a normal four to five second cycle. The lung is invested with elastic fibers which deflate the lung to varying degrees, at the close of the positive pressure period. The positive pressure period is initiated by the inhibition of the diaphragm and intercostal muscles. Though the diaphragm is fixed to the vertebrae in the area of the central tendon during a coughing episode the stomach rises towards the esophageal hiatus with a water-hammer effect. This thrust on a weak or malformed hiatus is the cause of hiatal and parahiatal herniations.

Evans'[3] discussion on "Sliding Hiatal Hernia" has brought out some interesting anatomical findings of the lower esoph-

agus, as seen on barium studies. Here we see a diagrammatic representation of the lower esophageal segment and esophageal hiatus. Dr. John A. Evans has kindly given me permission to use his illustration.

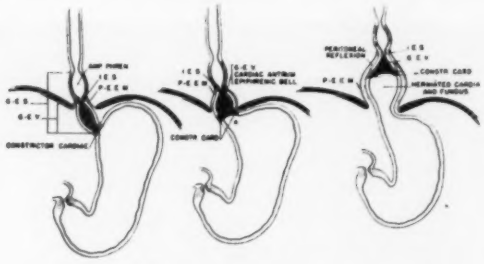

Fig. 1.

G.E.V. Gastro-esophageal vestibule-cardiac antrum.
G.E.S. Gastro-esophageal segment of expulsion.
I.E.S. Inferior esophageal sphincter.
P.E.E.M. Phreno-esophageal elastic membrane.

1. Relationship of the normal hiatus. Black area is the gastro-esophageal vestibule.

2. Relationship of the so-called hiatal insufficiency. The blacked-in segment of the esophagus lying in and partially above the hiatus, represents the distended gastro-esophageal vestibule, cardiac antrum or so-called epiphrenic bell of Anders and Bahrmann. It is this arrangement that is most difficult to interpret during Roentgen examination and must be differentiated from a small sliding hernia. During the roentgenoscopic phase of the examination, this is frequently possible only by demonstrating the integrity or incompetence of the cardiac sphincter.

3. Diagram of sliding hiatal hernia in frontal plane showing the relation of the various structures.

From this excellent diagrammatic representation it is clear to see how any thinning or malformation of the esophagus hiatus and intra-thoracic and intra-abdominal pressures makes possible hiatal changes by the dilatating action of the gastro-esophageal vestibule. In similar manner a para-esophageal herniation may occur through and along the hiatal connective tissue of the diaphragm.

Esophagitis, or better reflux esophagitis, occurs only when the sphincter is insufficient, and free hydrochloric acid is present. This condition has been attributed to inflammation and vagal and phrenic interruptions. It is the purpose of this paper to present two different types of cases to suggest another or newer cause of reflux esophagitis.

This is the case of an adult with a left pneumonectomy for a benign adenoma of the left bronchus. Postoperative course was uneventful. This was done four and one-half years ago, and at the time of operation there was no interruption of the vagus or phrenic nerve. For the past two years the patient

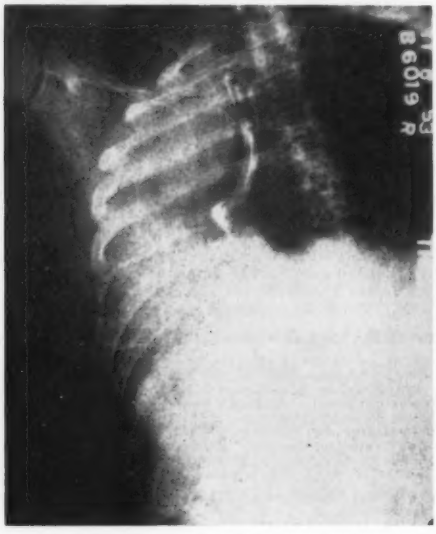

Fig. 2.
P. A. Film of Chest.

has had progressive episodes of heartburn and retro-sternal distress, which were relieved by medical treatment. Examination by X-ray shows this interesting picture of the esophagus, stretched, angulated and fixed to the latero-posterior parietal pleura by fibro-thorax. Esophagoscopy was not attempted. The lateral angulation and stretching has pulled the esophagus in such a way that the sphincter is no longer in a plane, but has incompetent abutting of the sphincter muscle; rather it is a lipping action, allowing some seepage of gastric contents.

The second case is that of a 40-year-old white female with a four-year history of scleroderma of the esophagus, proven by biopsy. For the past eight months she has had regurgitation,

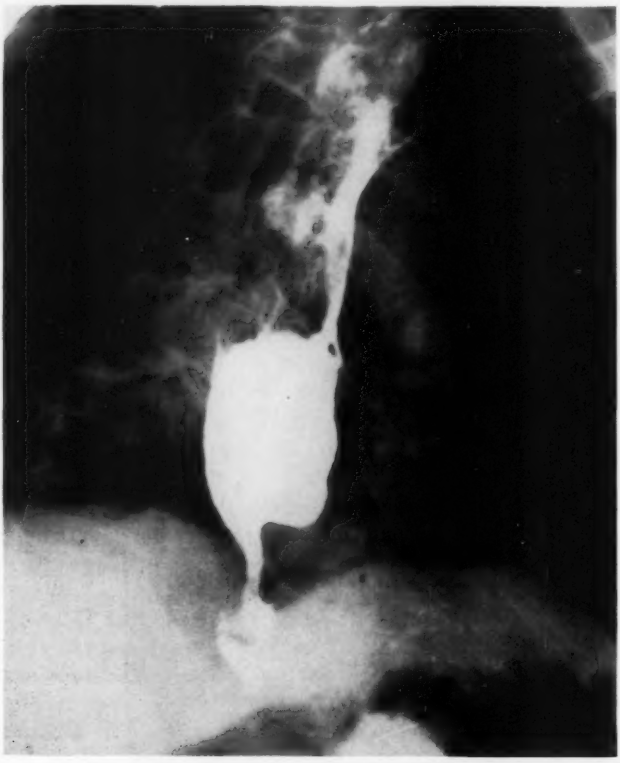

Fig. 3.

burning sensation and difficulty in swallowing. The stomach had free hydrochloric acid. Fluoroscopy failed to reveal any peristalsis; there was fixation of the musculature and sufficiency of the sphincter.

This picture shows the scleroderma taken four years ago, showing narrowing of the esophagus and dilatation of the proximal esophagus. A follow-up picture shows the recent presence of an ulcer caused by the esophagitis.

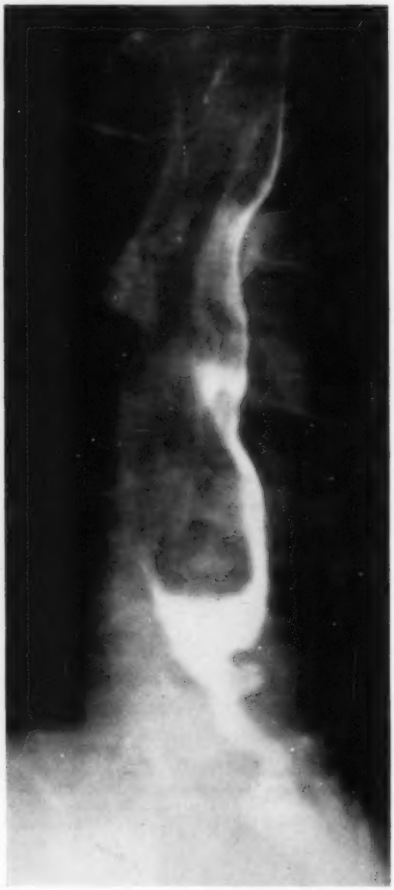

Fig. 4.

These two varied cases have developed esophagitis secondary to a lesion proximal to the lower esophagus, in each of which there was no interruption of the vagus or phrenic

nerve. It is difficult to show the pathology causing esophagitis before the patient comes to us with symptoms referable to the condition. Reflux esophagitis occurs only when the sphincter is insufficient and free hydrocholic acid is in the stomach. In these cases we have seen, the sphincter was made insufficient by traction in the man who had a pneumonectomy, and in the woman who had muscular displacement from a sclerotic lesion causing sphincter insufficiency.

It is difficult to imagine that a generalized inflammation of the sphincter area would cause reflux esophagitis, since the edema would be equal throughout this area. It seems more logical that lesions such as a tumor mass, an eroding carcinoma, a granuloma, or fibrotic lesion would cause distortion of the sphincter plane, aside from proximal lesions.

Concering the problem of esophageal diverticula, especially the epiphrenic type, Putney, et al.,[4] have brought out several etiological suggestions. In considering the non-traction type, it is interesting to note the work done by Strauss and Lubitz,[5] showing the glandular structure of the stomach, cardia and esophagus. Unlike the stomach and cardia, the esophageal glands are racemose and are found beneath the muscularis mucosa. This could cause a weakness in the esophageal wall, and any increased intra-esophageal pressure over a period of time would cause a herniation of the wall and form a diverticula, or diverticulae. Dilatations of these glands are common, and at times reached cystic proportions.

SUMMARY.

Problems of the lower esophagus have been presented on the basis of anatomical changes of the esophagus, diaphragm and cardia. The problem of reflux esophagitis has been considered, and believed to be caused by alterations of the sphincter plane, by local and proximal lesions. It is suggested that diverticula of the esophagus is caused by the glands being under the muscularis mucosa and causing weakness of the wall.

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CLINICAL OBSERVATIONS ON END-ORGAN DEAFNESS.*

A Correlation with Cochlear Anatomy.

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The concept that all cases of nerve deafness must inevitably progress on a predestined course is no longer tenable. Recent observations by Hilger, Goltz[1] and Williams[2] suggest that there is hope of salvage in some instances, if cases are individualized and etiological aspects are considered on a physiological basis, before irreversible changes occur in the inner ear. Pathological changes in the cochlea, which result in hearing impairment, gradually become irreversible. Therapeutic failures in the past have resulted from attempting to reverse advanced cochlear pathology, and relying solely upon the use of drugs for which the pharmacological effect was at best somewhat uncertain, as far as the ear was concerned.

Exact knowledge concerning the histopathology of early endorgan deafness is very limited, because the organ of Corti undergoes lytic changes soon after death; furthermore, current histologic technique alters the cochlear structures—in particular, the normal contact of the tectorial membrane with the hair cells and their supporting structures. Recent studies by Hilding[4] have confirmed the opinion expressed by Shambaugh, Sr.[3] in 1907, that the tectoriál membrane in the living state is attached to the surface of the organ of Corti. The possible clinical significance of this relationship has been discussed by Hilding[5] and will be further considered here.

In this discussion some cases will be cited; first, to illustrate significant improvement in auditory threshold, believed to have resulted from therapeutic measures. Second, some

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possible correlations between these clinical observations and known anatomical facts, together with theoretical considerations will be considered.

Bone conduction threshold and tuning fork tests were done on all cases, and established a diagnosis of nerve deafness in each. Only air conduction thresholds are shown in order to simplify the charts. The Eustachian tubes were inflated and found to be patent in all cases. These particular cases were selected because they represented a cross-section of the clinical problems encountered in managing the phase of otology under discussion.

Case 1. K. D., Age 20. Tinnitus was first noted four months ago following a cold. It was more noticeable in the right ear. Indefinite vertigo had been present for six weeks. A feeling of pressure in the ears and an increase in tinnitus preceded the episodes of vertigo. The radio sounded hollow and out of tune and had an echo-like quality. There were numerous gastrointestinal and chest symptoms which an internist concluded were due to autonomic instability. Patient smoked two to three packages of cigarets daily. There was no history of acoustic trauma. Dissatisfaction with the school he was attending and indecision as to a life vocation seemed to be the immediate basis of his psychosomatic disturbance.

Examination revealed normal tympanic membranes. Vestibular response to cold caloric stimulation was normal in both ears. He was given banthine and roniacol and advised to quit smoking. The tinnitus and vertigo gradually diminished and there was some subjective clearing of his hearing. It was believed that his inner ear upset was the result of his psychosomatic disturbance and excessive smoking. It will be noted in audiogram No. 2, Fig. 1, that there has been partial reversal of the audiometric notch in the right ear, but the left ear lesion had apparently progressed beyond the stage of reversibility.

Case 2. M. J., Age 27. A slight hearing impairment in the right ear had been present for two years following a suppurative otitis media. The patient was a surgical nurse, and in recent months there had been a lack of clearness in the left ear which caused some difficulty in understanding the mumbled words of surgeons. She smoked one package of cigarets daily. There was no tinnitus.

Examination revealed a large central perforation of the right tympanic membrane. The left tympanic membrane was normal. The audiometric curve reveals a notch at 2000, and a high tone loss in the left ear (see No. 1, Fig. 2). She was advised to quit smoking and was given roniacol by mouth. Three weeks later there was both subjective and audiometric improvement of hearing in the left ear (see Audiogram No. 2, Fig. 2).

Case 3. W. S. B., Age 39. Some hearing loss for high-pitched voices had been noted recently, especially in the left ear. There was some high pitched tinnitus, more noticeable in the left ear. A chronic postnasal discharge of variable degree had been present for several years. Five years previously the patient had had a temporary hearing loss following an upper respiratory infection. There was no history of acoustic trauma. He smoked about one package of cigarets daily.

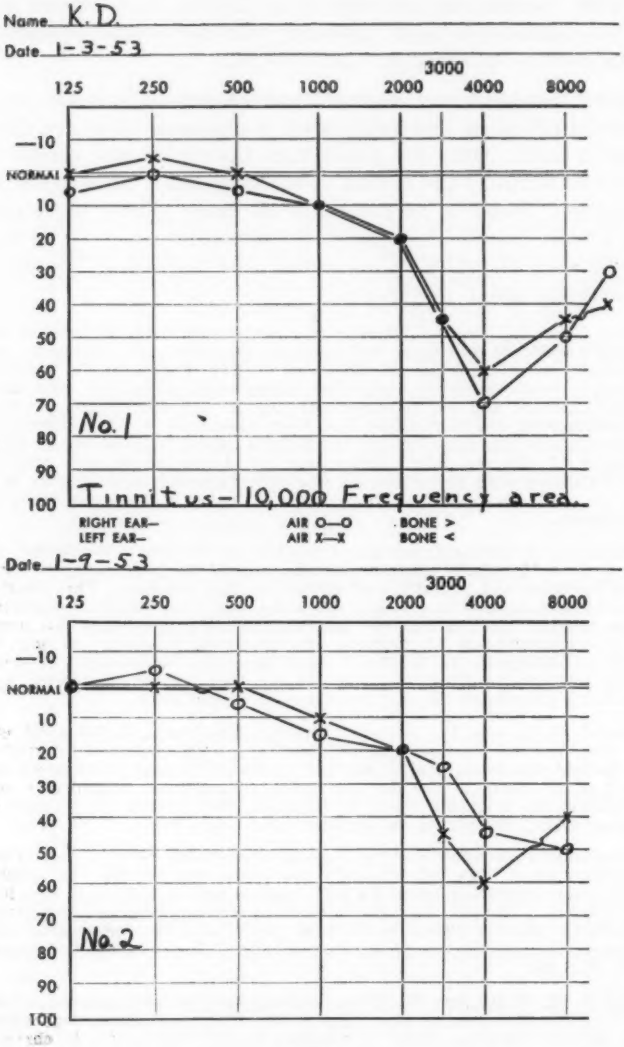
Name K. D.
Date 1-3-53
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
10 20 30 40 50 60 70 80 90 100
No. 1
Tinnitus—10,000 Frequency area.
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 1-9-53
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
10 20 30 40 50 60 70 80 90 100
No 2

Fig. 1.

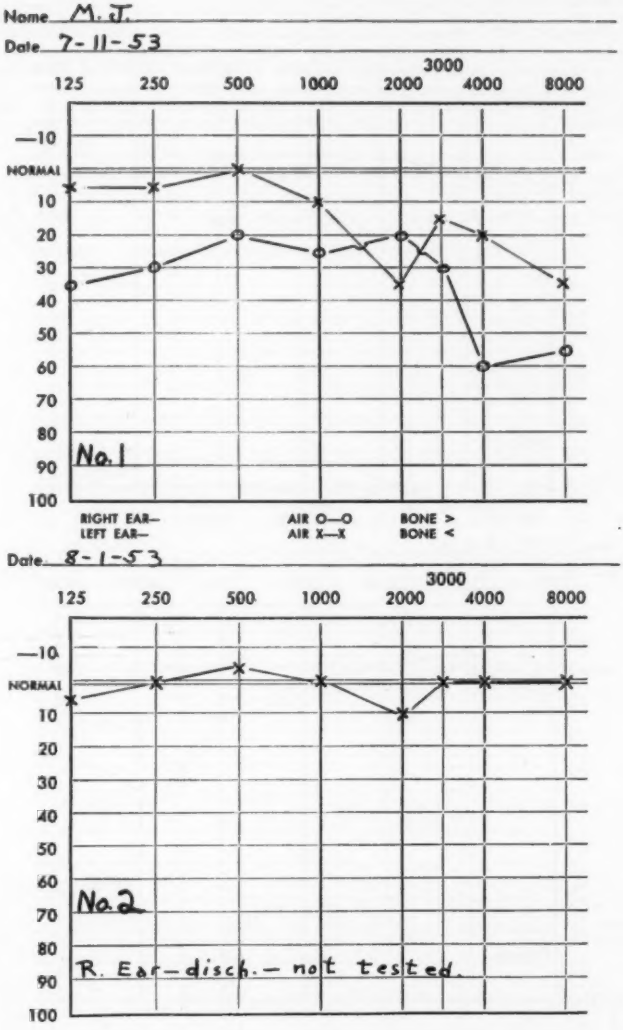
Name M. J.
Date 7-11-53
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
10 20 30 40 50 60 70 80 90 100
No. 1
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 8-1-53
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
10 20 30 40 50 60 70 80 90 100
No. 2
R. Ear—disch.— not tested.

Fig. 2.

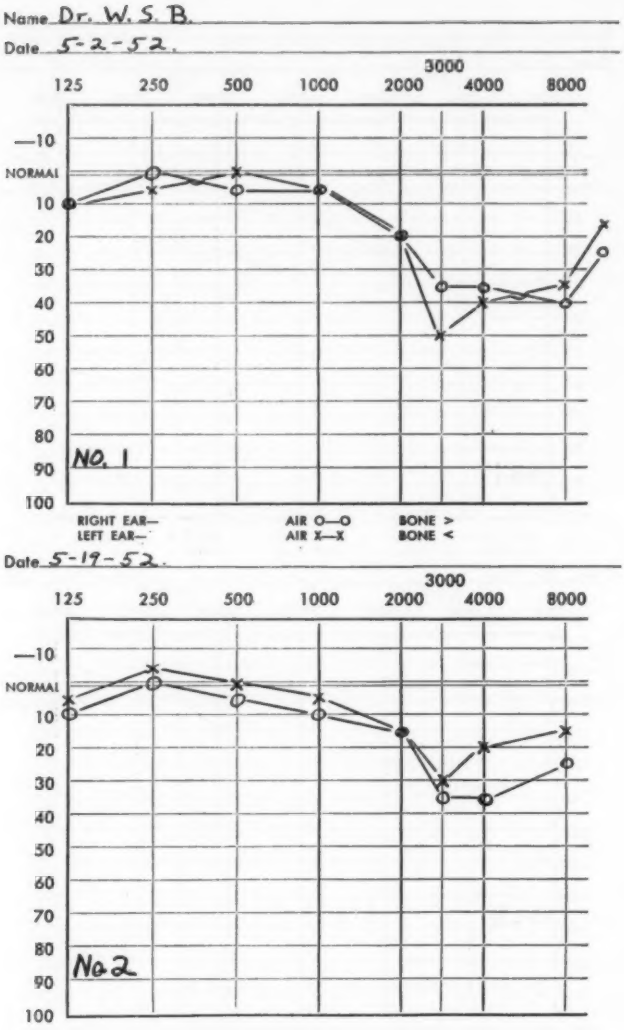
Name Dr. W. S. B.
Date 5-2-52.
125
250
500
1000
2000
3000
4000
8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
NO. 1
RIGHT EAR—
LEFT EAR—
AIR O—O
AIR X—X
BONE >
BONE <
Date 5-19-52.
125
250
500
1000
2000
3000
4000
8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
No 2

Fig. 3.

On examination both tympanic membranes were normal. There was a moderate amount of mucoid secretion in the nasopharynx, and some scattered follicles of lymphoid tissue were present. The sinuses were clear on transillumination. The initial audiogram shows a moderate high tone loss (see No. 1, Fig. 3). He was advised to quit smoking and was given roniacol. There was gradual subjective and objective improvement in hearing. A marked decrease in postnasal drainage was noted. The hearing improvement occurred chiefly for the high tones in the left ear (see No. 2, Fig. 3).

Case 4. F. G., Age 30. This patient was concerned chiefly with tinnitus following recent exposure to gunfire. He had noted tinnitus previously after hunting, but it had always ceased after a few days. He was not aware of any hearing impairment. There was a history of fall hay fever and perennial vasomotor rhinitis of moderate severity.

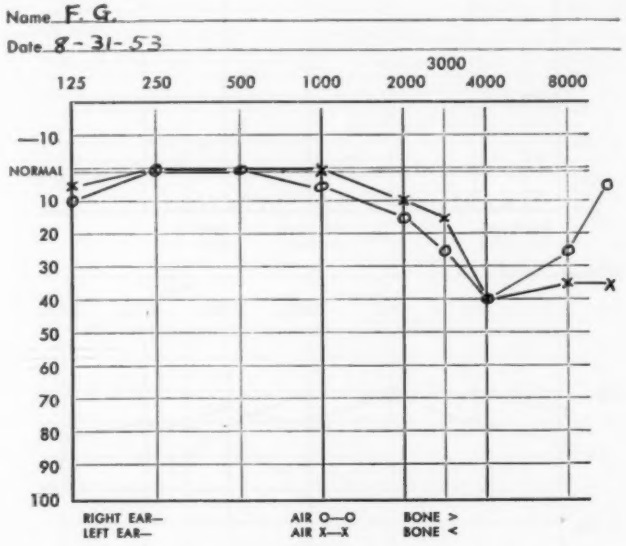

Fig. 4.

Both tympanic membranes were normal. Audiometric examination revealed a typical acoustic trauma notch at 4000 (see Fig. 4). He was advised to discontinue smoking and was given nitocinic acid by mouth. Ear plugs were provided for protection against future acoustic trauma. He did not return for follow-up study.

Case 5. T. J. N., Age 36. Attacks of vertigo, together with hearing impairment in the right ear, had been present for six weeks. These symptoms initially followed a rhinitis. Music seemed distorted. Intermittent tinnitus in the right ear was present.

On examination, the tympanic membranes were normal and the Eustachian tubes inflated readily. Audiometric examination revealed all of

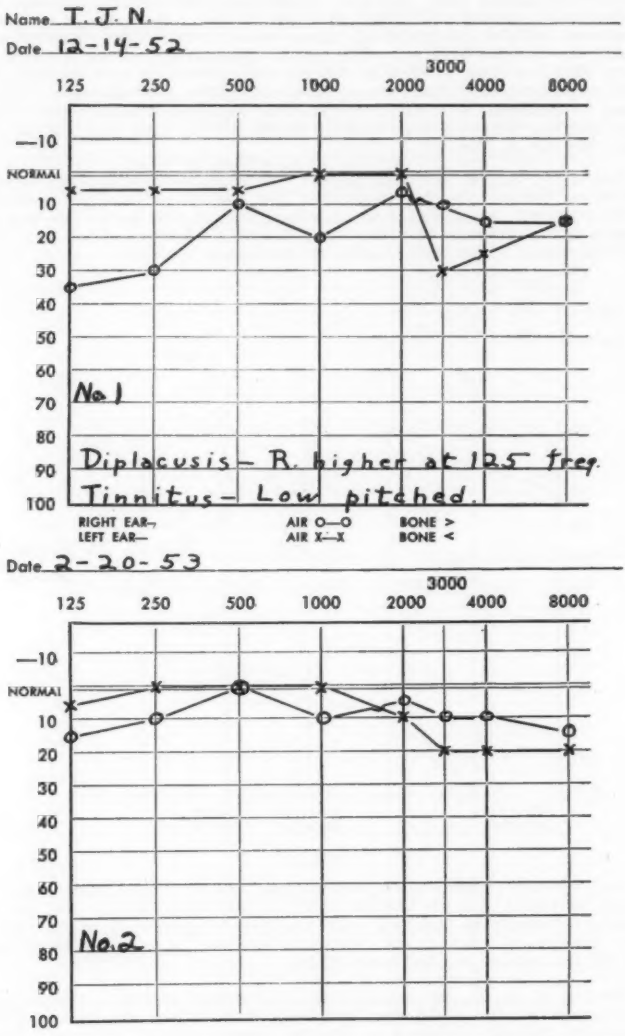
Name T. J. N.
Date 12-14-52
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
No. 1
Diplacusis – R. higher at 125 freq.
Tinnitus – Low pitched.
RIGHT EAR–
LEFT EAR–
AIR O—O
AIR X—X
BONE >
BONE <
Date 2-20-53
125 250 500 1000 2000 3000 4000 8000
—10
NORMAL
10
20
30
40
50
60
70
80
90
100
No. 2

Fig. 5.

the characteristic findings of an early endolymphatic hydrops (see No. 1, Fig. 5). The patient was placed on a low salt diet and given nicotinic acid and potassium chloride by mouth. There was a gradual decrease in tinnitus and vertigo, and the hearing gradually returned to near normal (see No. 2, Fig. 5). The diplacusis disappeared as the hearing improved.

Case 6. N. C., Age 27. Tinnitus and deafness were first noted in the left ear four years ago. More recently there had been deafness in the right ear. There was some fluctuation of hearing at the onset, but none in recent months. Considerable mucoid postnasal discharge occurred in the morning. An episode of sneezing was experienced once or twice a week. Radium therapy to the nasopharynx had been applied with no benefit. The patient had been advised to obtain a hearing aid, but had not done so. Numbness of the fingers was frequently noted when doing such work as knitting.

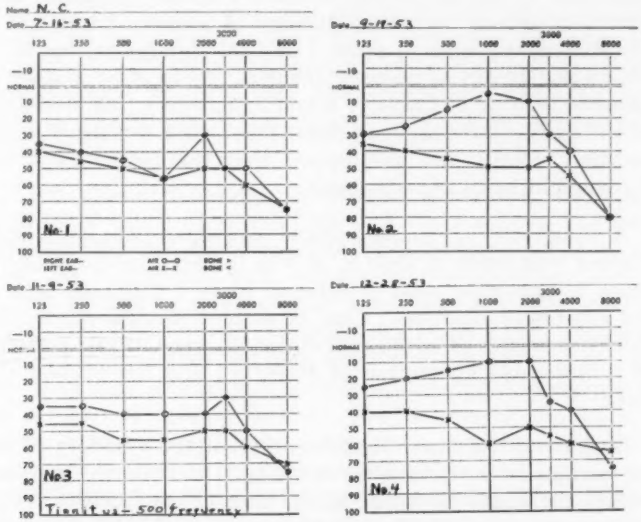

Fig. 6.

On examination, both tympanic membranes were normal and the Eustachian tubes inflated without difficulty. There was an excess of mucoid secretion in the nasopharynx. A smear of this secretion contained many polymorphonuclear cells and bacteria but no eosinophiles. The sinuses transilluminated clearly. The initial audiogram revealed a moderate degree of loss (see No. 1, Fig. 6). Speech tests revealed poor discrimination, especially in the left ear, and the narrowed dynamic range characteristic of recruitment.

The patient was advised to quit smoking and was given nicotinic acid by mouth. On this treatment there was a gradual improvement in hearing in the right ear and a noticeable decrease in postnasal drainage during the warm dry summer months (see No. 2, Fig. 6). Smoking was

not entirely stopped but was decreased by about half. In the fall there was again a decrease in hearing (see No. 3, Fig. 6). The increased stress of weather variations and attending night school, in addition to her daily work as a medical technician perhaps contributed to this hearing decrease. Allergic studies cast some suspicion on milk. It will be noted that there was no improvement in hearing in the left ear, which was initially involved before the right. Evidently the changes in the left ear had progressed to a state of irreversibility. The last audiometric test is shown in No. 4, Fig. 6. The improvement in the right ear followed a more rigid avoidance of milk.

COMMENT.

The question might be raised as to whether the tonal dips and audiometric configurations in Cases 1, 2 and 3 represent pathology in the end-organ or in the spiral ganglion. It is generally agreed that the pathology of acoustic trauma is in the end-organ. Case 4, from a clinical standpoint, would be considered as one of acoustic trauma. There is no history of acoustic trauma in Cases 1, 2 and 3; however, the similarity of the audiometric curve of these three cases to Case 4 would certainly justify the assumption that the pathology in all three might be in the end-organ.

A diagnosis of endolymphatic hydrops (Meniere's syndrome) was made in Case 5. It was included in this study in order to illustrate what is believed to be a different type of the end-organ deafness. Case 6, from a clinical standpoint would be a case of cochlear hydrops, in view of the presence of considerable fluctuation of threshold and low-pitched tinnitus.

In most of the cases cited there was either a history of the ear symptoms starting after some type of rhinitis, or of excessive postnasal discharge at the time of examination. This relationship gave rise to the older concept of catarrhal deafness; however, rather than representing a cause-effect relationship between the nasal symptoms and ear disturbance, the nasal and ear symptoms may represent a simultaneous clinical expression of a basic neurovascular and neurosecretory disturbance in two areas of the autonomic and vascular distribution.

For this reason, it is necessary to consider the therapeutic approach beyond the confines of the nose and nasopharynx, and analyze each case individually, as to the many environ-

mental and intrinsic factors which contribute toward the basic physiological disturbance.

An accurate evaluation of therapy in hearing impairment, believed to be due to end-organ changes, is extremely difficult because in some instances there is early reversibility even though no therapy has been applied. This is readily understood when we consider the diversity of etiological factors which may vary from emotional and psychic stress to definite infection in the upper respiratory passage. These etiological factors may singly or in combination express themselves in a final common pathway of arteriolar-capillary-venular dysfunction with consequent local electrolyte and nutritional disturbances involving the structures concerned in inner ear physiology. The physiological and hypothetical aspects of this problem have recently been comprehensively and aptly covered by Hilger[1] and Williams[2] and need not be considered in detail here.

Loch[6] studied tonal dips in 1365 school children, and found dips in 15 per cent of the boys and 5 per cent of the girls. He classified the dips into persistent, temporary and recurrent. Persistent dips were noted most frequently at the 4096 frequency and temporary ones were more often observed in the 10,321 area. About two-fifths of all dips were permanent. We can assume that the temporary dips disappeared because the etiological factor was corrected, either spontaneously or as a result of definite therapy. The permanent dip must be accepted as an irreversible lesion, or as an instance in which appropriate therapy was not applied. This study would certainly suggest that a tonal dip must not always be accepted as being inevitably permanent until a reasonable attempt has been made to uncover possible etiological factors and until indicated therapy has been applied; however, this must not be construed as an invitation toward unreasonably long and expensive treatment. Destructive surgical attack on questionable foci of infection is certainly to be condemned.

Wever[7] studied the Hopkins temporal bone sections concerning the problem of tonal dips and found no consistent pathological changes; however, he stated that "failure to find a lesion in a particular region does not signify that the tissues

there were operating in a normal manner. There is no certainty that functional impairments are revealed by the histological technics used."

He observed that deafness in general, especially abrupt high tone loss, was noted more often in men. In speculating as to the basis of occurrence of tonal dips more frequently in the 4096 area, he considered two possibilities; first was the fact that the 4096 frequency lies in the region of resonance of the ear; hence this area is probably subjected to more mechanical disturbance. Second, he believes this area to be subject to more autolysis postmortem, possibly as a result of more susceptibility to physical breakdown in life.

An interesting feature of the anatomy of the cochlea is the constriction of its bony wall at the approximate region of perception for the 4096 tone (see Fig. 7). Larson[8] has listed this as one of the possible reasons why the initial loss secondary to acoustic trauma in general is greatest for the 4096 frequency. It is obvious that the sound pressure incidental to each cycle with a frequency lower than 4096 must pass through this "bottleneck." This fact, together with Wever's statement that the region of resonance of the ear is at the 4096 frequency, would mean that the cochlear structures in this area must be subjected to considerably more mechanical stress than in any other area.

There has been a marked renewal of interest in the tectorial membrane since the recent studies made by Hilding.[4] The usual textbook picture showing the membrane separated from the hair cells and their supporting structures is an artefact of tissue fixation and dehydration. In fresh specimens the tectorial membrane is very intimately bound to the organ of Corti. Hilding has incorporated this relationship into a theory of sound perception. Borghesan[9] has furnished further histological evidence which substantiates Hilding's findings and goes on further to state that he believes that the hairs from the hair cells are continuous with and, therefore, constitute the fibrils of the tectorial membrane. The fibrils are imbedded in an amorphous mass of gelatinous substance, which in the living, is almost fluid because of its high water

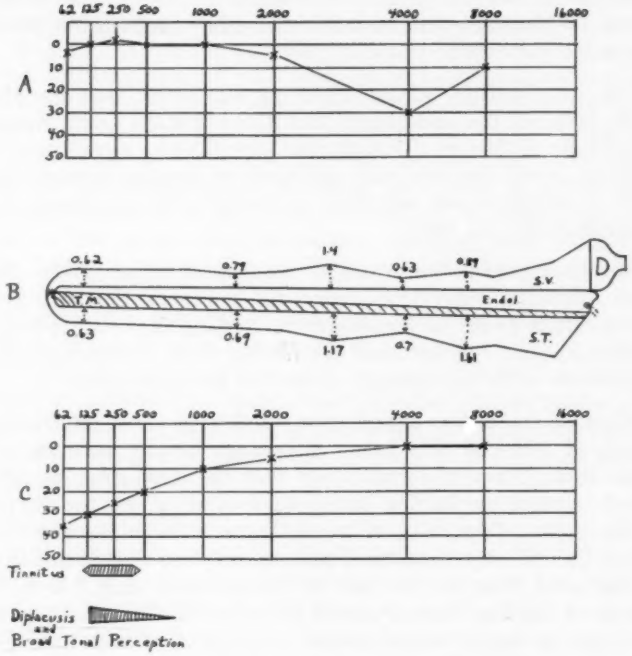

Fig. 7.

A. An audiometric chart modified so that the position of each frequency on the chart corresponds to its approximate area of tonal perception in the cochlear sketch below in B. The audiometric curve illustrates a typical 4000 notch.

B. This is a sketch of cochlear dimensions taken from Fowler's loose-leaf "Medicine of the Ear." I have modified the sketch by inserting the endolymphatic duct and the tectorial membrane. The relative size of the tectorial membrane and the endolymphatic duct are schematic from descriptions in the literature, and must not be accepted as representing exact relationships. Points to be noted are the constriction of the cochlea at the 4000 frequency area and the gradual increase in size of the tectorial membrane from the base to the apex.

S. V.—Scala vestibuli.
Endol.—Endolymph.
H.—Helicotremma.
S. T.—Scala tympani.
T. M.—Tectorial membrane.

The numbers on the outside indicate the cross section area of the adjacent scala vestibuli and scala tympani in MM.²

C. An audiometric chart arranged as in A with a curve characteristic of cochlear hydrops. The tinnitus, diplacusis and broad tonal perception are indicated as occurring in the low frequency area.

content. He believes that the shrinking of the tectorial membrane incidental to the usual histological preparation tears the hairs just above the cell itself.

The interposition of the tectorial membrane between the main body of the endolymph and the organ of Corti would necessitate diffusion of endolymph through the membrane in order to reach the hair cells and their supporting structures. Such a function was ascribed to the tectorial membrane by Richenbacher[10] in 1901.

Embryologically the tectorial membrane arises from the same epithelial ridge that forms the organ of Corti (Prentiss[11]). This might be considered as lending further credence to Borghesan's concept that the fibrils of the membrane are continuous with and actually a part of the hair cells.

What, then, is the significance of this possible additional aspect of tectorial membrane physiology to the question of tonal dips? Since it seems likely that the 4096 area is subjected to more mechanical stress than other areas, the fibrils at the point of junction of the main tectorial mass and the hair cells will be traumatized and possibly torn more readily in this area than in the rest of the cochlear duct. A mild degree of fibrillar damage could be reversible but perhaps a more severe degree would not be. The effect of such damage would be twofold: first, there would be interference with the mechanics of sound perception in this area; second, the nutrition of the hair cells would be impaired.

If this point of constriction is significant, then variations in the degree of constriction could account in part for variations in susceptibility to acoustic trauma and non-traumatic early physical deterioration in this area, with a subsequent tonal dip. Any minor disturbance in nutrition of the organ of Corti in general, which otherwise might not lead to impairment of cochlear perception might, in the presence of a greater degree of cochlear constriction in this area, result in definite localized diminution in hair cell perception.

THERAPY.

The rationale of therapy applied to the cases cited earlier is based largely on the concepts expressed by Hilger and

Williams. A small but definite percentage of individuals has an unquestioned harmful physiological response to the effects of smoking. It has been routine to advise patients to discontinue smoking during the immediate postoperative period after fenestration. On resuming smoking many of the patients note a definite increase in vertigo during the smoking of each cigaret. The fact that not all patients experience this reaction substantiates the idea that there is a wide individual variation in tolerance to nicotine. I believe that in most of the cases cited previously, smoking was a significant factor in the etiology of disturbed inner ear physiology.

Vasodilating therapy is given on the presumed presence of vasospasm of some portion of the inner ear vascular tree. Psychological stress in the form of interpersonal emotional problems, business or other responsibilities exceeding the individual's normal capacity, frustrations, etc., must be taken into consideration in proper perspective in the overall management of these cases.

The excessive use of caffeine in any form may be a contributing factor in etiology. The individual who fortifies himself with a stimulant several times a day whenever his energy lags may be "whipping a tired horse" and inviting eventual imbalance of the intricate and sensitive autonomic system.

The role of undue physiologic fatigue in initiating disease in general is too well-known to require further comment; however, the importance of fatigue to the point of exceeding one's physiological limits is too often neglected in counseling our patients in this age of stimulants and sedatives, histamine and antihistamines, vasoconstrictors and vasodilators, etc. Unfortunately, too many patients insist on pursuing ways and habits of living which for some can eventually lead only to a state of hopeless autonomic confusion. They seek to obtain refuge from their ultimate fate of physiological disorganization by consuming multiple boxes of pills rather than by reestablishing a more hygienic mode of life.

Allergy is another aspect of etiology which must be given proper consideration. Jordan[12] has reported a few instances of tinnitus due to proven food allergy. Tinnitus is frequently

the earliest symptom of impending cochlear deterioration. The allergic problem is definitely interwoven with that of autonomic dysfunction, as is illustrated by Case 6.

The relationship of acetylcholine release or formation incidental to trauma in a closed space is of theoretical interest. Increased free acetylcholine has been found in the spinal fluid of animals subjected to experimental cerebral trauma.[13] On the basis of this study, large doses of atropine have been used with some benefit in treating selected cases of cerebral trauma in humans.[14]

If the concept of mechanical stress incidental to the 4096 cochlear constriction is valid, it is conceivable that repeated excessive local liberation of acetylcholine could increase capillary permeability sufficiently to initiate a cycle of arteriolar-capillary-venular dysfunction. This would eventually predispose the area to autonomic imbalance and its consequences, whenever any one or a combination of several of the previously discussed etiological factors became operative.

In other words, a small segment of the vascular bed, which has previously become conditioned to respond in a stereotyped manner to local trauma, under conditions of general stress may react to a degree that proves detrimental to the local structures. The individual capacity to withstand stress varies greatly, and may account for the fact that the same etiological factors which will produce inner ear disturbances in one individual are well tolerated by others.

Endocrine balance is intimately related to autonomic function and must be given proper consideration, as indicated by the history and clinical picture.

ANATOMICAL CONSIDERATIONS.

The possible significance of the constriction of the bony wall of the cochlea in the region of perception for the 4096 frequency has already been discussed. The degree of variability of this constriction needs to be studied further. Minor disturbances in the integrity of the tectorial membrane may prove to be important in explaining the cause of perceptive lesions for which to date there has been no visible anatomical

basis on microscopic study. Once the physical continuity of the tectorial membrane, with its attachments, has been disrupted in a small area, continued mechanical stress will undoubtedly spread this disruption in much the same manner as a strong wind would extend a small tear in a sail.

The inner ear disturbance in Case 5 is due to endolymphatic hydrops. The cochlea of the involved ear (right) presents all the early findings characteristic of this condition, *i.e.*, greater loss for the lower frequencies and a coarse broad character of tonal perception for the lower frequencies. It will be noted that the early symptoms all occur in the apical area of the cochlea (see Fig. 7-C). If the deafness continues to progress, the loss eventually involves the entire audiometric scale.

Examination of the relative dimensions of cochlear structures reveals that the tectorial membrane is much larger in the apex than in the base. The scala media, on the other hand, is slightly smaller in the apex than in the base. Unfortunately, current histological techniques do not permit any conclusion as to the state of the tectorial membrane in endolymphatic hydrops before the artefact of fixation and dehydration is produced.

The fibrils of the tectorial membrane are imbedded in an amorphous mass of protein-like substance, which is no doubt altered in physical characteristics, and possibly size, as a result of the altered character of the endolymph in an ear with hydrops. In all probability, any appreciable edema of the membrane would change its physiological response to sound more in the apex than in the base because of the greater size of the membrane in the apex. Shambaugh, Sr.,[3] first suggested that the tectorial membrane changes mights account for such cochlear symptoms as diplacusis and some instances of tinnitus. Mygind[15] has more recently mentioned tectorial membrane changes in labyrinthosis.

Cook[16] has noted that patients who clinically have cochlear hydrops are unusually susceptible to acoustic trauma. Relief of the hydrops by medical measures enabled a number of such individuals to return to working in a noisy environ-

ment. We might speculate that the edema of the tectorial membrane created an unusual tension on the hair cells insertion into the membrane. As the edema subsided this abnormal relationship was corrected, and as a result greater tolerance to intense sound returned. Cook's observations might suggest some relationship between recruitment and changes in the tectorial membrane.

The question as to what happens in the fluid-containing spaces within the organ of Corti in cochlear hydrops still remains to be answered. Another question posed is why, in the presence of seemingly similar etiological factors, does the cochlea in one individual respond with a clinical entity interpreted as cochlear hydrops, and another with a tonal dip? The answer may very well lie in variations in anatomy such as areas of cochlear constriction and tectorial membrane size. General individual physiological deviations involving capillary permeability and electrolyte balance may predispose some ears to an edema-type of response in the presence of autonomic dysfunction. Pearlman, et al.[17] have recently pointed out the limitations of our present knowledge concerning the relationship of electrolyte balance and Meniere's disease.

SUMMARY.

1. Several cases are cited in which reversal of an early end-organ deafness was observed. The therapeutic approach applied to these cases was relatively simple.

2. Failure of both ears to improve indicated that the lesion had progressed to a stage of irreversibility in one ear.

3. Enthusiasm for the efficacy of a given therapeutic measure must always be tempered with the possibility that there is in some instances a spontaneous reversal of the lesion.

4. A broad physiological approach to a wide range of etiological factors is perhaps the most important aspect of the general problem of early nerve deafness.

5. Theoretical correlations have been made concerning the possible relationship of some features of cochlear anatomy and clinical observations on nerve deafness.

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THE ANTIBIOTICS AS AN ADJUNCT IN THE TREATMENT OF OTOLARYNGOLOGIC DISEASES.*

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In recent years the antibiotics have been introduced into our armamentarium to combat infection as a *deus ex machina*, so sensational has been their effectiveness. It is a precarious venture to enter into a primary dissertation on the antibiotics before an audience of this caliber. I must, therefore, limit my comments to a few remarks in order to refresh our memories and to bring us onto a common ground for purposes of discussion. The picture changes so rapidly that categorical statements are futile, since a certain amount of confusion and lack of sequence is inevitable.

From the very comprehensive treatise on *Antibiotic Therapy*[1] it may be of historical interest to quote the following paragraph:—

"The term 'antibiosis' probably was used first by Vuillemin[2-1] in 1889 to describe what we consider to be 'the survival of the fittest', whereby a creature destroys the life of another to preserve its own. The use of the word 'antibiosis' to describe microbial antagonism was adopted by Marshall Ward ten years later.[3-1] It was not, however, until 1942 that Wakeman[4-1] proposed the use of the term 'antibiotics' to define those chemical substances of microbial origin which possess anti-microbial activity. With the increasing search for new antibiotics, this term has been enlarged to include not only chemical substances of microbial origin, but also those of plant and even animal tissues. In any case, the word antibiotic has now become firmly established, not only in the

* Read at the meeting of the Middle Section of the American Laryngological, Rhinological, and Otological Society, Inc., St. Louis, Mo., January 18, 1954.

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minds of scientists throughout the world, but in the lay mind as well, through the tremendous amount of publicity these drugs have received during the past ten years. Notwithstanding the fact that the term antibiotic is misused in some instances, and that it may not be the best word to define this important group of substances, it is unlikely that it can be displaced by another of perhaps greater merit, such as 'antimicrobial,' 'antagonist' or 'bacteriostat,' because it has become so firmly fixed in both the scientific and lay literature."

Under the pressure of our daily routine we may be prone to accept the antibiotic as an omnipresent umbrella to tide us over the worst cloudbursts of our clinical storms, with the hope that we may avoid the devastating inundations of the more serious and intractable infections that bring havoc to the suffering patient. It may be, that at times, we do not give sufficient thought to the serious reactions of an antibiotic in our efforts to accomplish the primary salutary effects.

It is well to jog our memories to recall the basic principles of resistance to infections as predicated by the antibody reaction of the individual. The mechanism underlying recovery in all infections is essentially anti-bacterial in character, brought about by the development and the actions of specific anti-bacterial antibodies, together with the activity of the phagocytic cells. This bio-chemical response to infection is still recognized as the all-important factor in preventing and limiting bacterial invasion.

It is not amiss to state that only too frequently this balance is disturbed by the premature administration of antibiotics, thus depriving the body of an adequate stimulus for the formation of antibodies and resulting in a pseudo-control of infections, with subsequent exacerbations because of a temporary and inadequate bacteriostasis of pathogenic microorganisms. The cardinal principle that resistance to infection is natural, or/and acquired, and that acquired resistance is the result of minimal or manifest infections, is an accepted theorem.

In otolaryngology, the antibiotics in common usage have been Penicillin, Aureomycin, Terramycin, Chloromycetin, Streptomycin, Dihydrostreptomycin, and recently Ilotycin.

Penicillin is primarily active against the gram-positive group of organisms, either as a bacteriostatic or a bactericidal agent. It is also active, but less so, against meningococci and gonococci in the gram negative group, while hemophilus influenzae is resistant to it.

While it was originally stated[1] that only certain strains of staphylococci became resistant to penicillin, Romansky[5] has brought out the fact that in the past few years investigations have shown that there is also occurring an increasing resistance of hemolytic staphylococcus aureus, hemolytic staphylococcus albus, non-hemolytic and alpha-hemolytic streptococcus (viridans).

Streptomycin and Dihydrostreptomycin. The chief concern of otolaryngologists in these antibiotics lies in their toxic reactions upon the internal ear, on the cochlear as well as the vestibular divisions of the VIIIth nerve. Streptomycin and dihydrostreptomycin affect both gram-positive and gram-negative organisms as bacteriostatic as well as bactericidal agents; however, the mycobacteria is the most important group of organisms acted upon by these drugs as bacteriostatic agents. They are ineffective against fungi, viruses and rickettsia. Hemophilus influenzae and pertussis are sensitive to their action. While these are drugs of supposedly low toxicity, para-aminosalicylic acid is being successfully used in combination to delay the growth of resistant strains of the tubercle bacillus and enhance the action of streptomycin.

Aureomycin, primarily bacteriostatic, is bactericidal in high concentrations. It affects both gram-negative and gram-positive organisms, also certain large viruses and rickettsia. These organisms do not easily develop resistance to the drug.

Terramycin. This drug has practically all of the characteristics of aureomycin, with the advantage of being less toxic and causing fewer and less severe side reactions.

Chloromycetin until recently was considered to be one of the most effective and least toxic of the broad-spectrum antibiotics at our disposal; however, in 1950 and subsequently, reports have appeared in the literature of blood dyscrasias, specifically aplastic anemias, which occurred following the

administration of chloromycetin over varying lengths of time. Among these, several fatal cases were reported, one within two days of the first administration of the drug.[6] These dire results might have been anticipated, since Smadel in 1949[7] had sounded a warning that the presence of the nitrobenzene radical in chloromycetin constituted a "potential danger as a hematopoietic toxin." As a consequence, the use of this drug has now been practically discontinued.

Despite the unquestionable effectiveness of the antibiotics, their uses have been curtailed by the many toxic reactions that have ensued. Such reactions we have all seen. We know that penicillin may cause a black tongue, a glossitis or a stomatitis, various degrees of urticaria; also more serious anaphylactic and allergic reactions, or even a true Herxheimer reaction. These distressing and alarming sequelae may develop after one or many administrations, occur immediately, or two or three weeks after its use is discontinued. Severe reactions may be averted in patients who have been given penicillin previously, by prescribing it orally; this avoids the immediate "build up" that may follow intra-muscular injections, especially if coupled with antihistaminic therapy.

The toxicity of the broad-spectrum antibiotics is usually manifested by gastric symptoms, nausea, at times vomiting, and diarrhea. The diarrhea and anal or vulvar pruritis are the more frequent and the more severe results of sensitivity. The diarrhea is said to be caused by the bactericidal effect of the antibiotics on the normal flora of the intestinal tract and their replacement by the acid producing yeasts or fungi of the monilial family especially. Despite the frequently published reports that the incidence of this penomenon is an occasional complication, our experience has been to the contrary. In some instances it has been so serious that it has established a colitis and a pruritis ani that has persisted or recurred for two or three years afterward.

Recently it has been determined that some of the untoward side reactions may be avoided and greater effectiveness achieved by the synergistic action of one or more of the antibiotics, combined with each other, or with chemotherapeutic agents. This is notably true with penicillin and streptomycin,

dihydrostreptomycin, or with the triple sulfonamides, and the use of the broad-spectrum antibiotics with the triple sulfonamides. We are all familiar with such combinations which have been made available by many of the manufacturers of these drugs. In some instances these seem to exert a greater antimicrobial activity on the border-line strains of certain organisms, or on those which may have become resistant to one or other of the drugs given singly; however, if signs of sensitization are manifest one is at a loss to know which drug may be the offender, if not both drugs simultaneously.

The question of organisms developing resistance to the various antibiotics and chemotherapeutic drugs has become a realistic fact, as more and more of these drugs are being used, and more accurate *in-vitro* experiments reported.

Romansky[5] states that recently many more gram-positive and gram-negative organisms have shown an increased resistance to the broad-spectrum antibiotics, as well as to penicillin. This is especially true in regard to streptomycin, the use of which has not only activated many resistant strains of various organisms, but has caused an enhancement of growth of tubercle bacilli, according to Speedlove, et al.[8] This phase of antibiotic therapy must lead one to conjecture on the significance of the passage of certain pathogens from one individual to another. Such pathogens, carrying an increased virulence as the result of animal passage, and an acquired resistance to antibiotics because of previous exposure, necessarily or otherwise, may be instrumental in causing the intractable spread of certain infections in a community, even to epidemic proportions.

The intelligent use of antibiotics when carefully chosen and distinctly indicated, despite the known possibility of more or less serious side reactions, is a justifiable and calculated risk; however, the haphazard use of these drugs or their various modifications in solutions for intranasal medication, or for ear drops, or as lozenges or ointments, with the knowledge that sensitizations may be imminent, and with no assurance of benefit to the patient, is contra-indicated. Likewise, the routine administration of penicillin as a pre-operative adjunct when infection is not present, and as a protection against a

presumptive infection with gram-positive organisms, thus unnecessarily exposing the patient to serious sensitization, is no more rational.

It is a generally accepted fact that the administration of the antibiotics should be governed by the sensitivity of the infecting organisms to the drug. We are all familiar with the procedure of obtaining a culture of the pathogenic flora and determining their sensitivity by special cultural methods. The time lapse for such a report from the laboratory is usually four to five days, a loss of valuable time, which may occur in the most critical period of a patient's illness.

Realizing this deficiency we have been conducting experiments in the Department of Otolaryngology of St. Louis University at the Firmin Desloge Hospital Clinic, to determine whether it is feasible and accurate to obtain this same information in a shorter length of time.*

Our plan of procedure has been as follows:

1. Tryptose-broth, whole-blood-agar plates used.

2. Culture taken from patient with swab, which is twirled in 8 cc. of tryptose broth.

3. Blood agar plates (3) flooded with 8 cc. of inoculated broth, the excess being removed with sterile pipette.

4. Surfaces of these plates were then allowed to become comparatively dry, and the discs containing the antibiotics were placed on the surfaces with sterile forceps. The sensitivities were routinely run against the following antibiotics in the disc concentrations indicated:

Aureomycin	60 mcg.	30 mcg.	10 mcg.
Chloromycetin	60 mcg.	30 mcg.	10 mcg.
Terramycin	60 mcg.	30 mcg.	10 mcg.
Penicillin	10 μ.	1 μ.	0.5 μ.

5. The inoculated plates with the discs were incubated in an inverted position for 24 hours, and the zones of inhibition

* Funds for these experiments were obtained as a grant from the "Stuever Fund" of the Department of Otolaryngology of the St. Louis University School of Medicine.

measured with a millimeter rule, the width of the zone indicating the effectiveness of the inhibiting property of the antibiotic against the entire flora cultured.

6. The individual colonies were then picked and planted in 3 cc. tryptose broth, incubated for 24 hours, and replanted on blood-agar plates with the antibiotic discs in place as in the 24-hour plate technique. After another 24 hours of incubation the zones were noted, measured and compared with those found with the respective antibiotics on the 24-hour plates.

In this manner we were able to verify the accuracy of the rapid method for determination of the sensitivities of the organisms growing in symbiosis, compared to the slower method of the reactions of the organisms growing individually.

This survey extended over a period of 12 months. The cultures were taken from the throat, nose or ears of unselected patients in the Firmin Desloge Clinic. In a total of 250 patients: 70 per cent were throat cultures, most of which were taken as part of our routine procedure the day before a tonsillectomy; 17 per cent were from the ears, and 13 per cent from the nose.

The pathology present is not pertinent to this report, although the organisms found were non-pathogens, as well as pathogens, of nine different varieties.

In the 24-hour mixed cultures the zones of inhibition corresponded to those in the pure cultures in 237 instances, or about 95 per cent. The zones about the two weaker disc concentrations were considered more significant than those about the strongest concentrations. As a rule, the zones were about three-fourths as wide in the mixed cultures as in the pure cultures in similar antibiotics, and in the majority of the plates penicillin was the least effective inhibitor in the mixed cultures.

Of the mycins, chloromycetin usually created slightly wider zones than aureomycin and terramycin, which were about

equal. In the later months of the survey ilotycin was used, though not recorded here, and it was found to inhibit less strongly than aureomycin and terramycin.

The types of organisms inhibited corresponded in general to the accepted criteria for the individual antibiotics.

From these findings we felt justified in deducing that *in-vitro* sensitivity tests of the anti-microbial properties of aureomycin, chloromycetin, terramycin, and penicillin, against 24-hour plated cultures of the entire bacterial flora of a focus, is at least 95 per cent as accurate as such tests against the pure cultures, which require 72 to 96 hours to interpret, using the disc technique on blood-agar plates as specified above.

The advantages of the 24-hour method are obvious in clinic practice, whereby the antibiotic thus indicated may be given almost immediately, and modified at the end of the 72-hour period if it is found advisable to do so.

In reviewing the many disadvantageous factors which have developed in connection with the administration of the antibiotics we are forced to admit that they are not the "panacea" which they originally gave promise of being. The unpleasant, and at times critical, reactions cannot be ignored, even though they may be ameliorated by the use of the antihistaminic drugs. Likewise the antibiotic resistant organisms, which nullify the anti-microbial properties of these drugs, must be respected.

We as physicians must not lapse into the unpardonable attitude of regarding the patient as a "test tube." The progress of infection in relation to the resultant pathological picture in the sinuses, the larynx and the ears must be carefully observed and therapeutic measures modified or discontinued as the indications arise. I view with great alarm and the utmost apprehension the prospect of irreversible hyperplastic otitis media and future conductive deafness in children who have been given the antibiotics to control otalgia or otitis media without keeping the condition of the membrana tympani under close observation, performing a paracentesis when indicated, and also testing the hearing, as apparent cure of the otitis media ensues.

We have seen many patients with unresolved maxillary sinusitis causing recurrences and constitutional signs of focal infection resulting from inadequate or no local treatment in addition to routine antibiotic therapy.

Flagrant instances are occurring daily of patients "passing on" their antibiotics to other members of their families and friends to cure infections which they consider similar to their own. For this reason a patient should be told which antibiotic he is being given, the symptoms of the possible reactions, and cautioned not to take the responsibility of administering the medication to anyone else. This advice should be prefaced by questioning the patient about previous exposure to antibiotics, and the possibilities of his own sensitivity to them should be carefully discussed.

It is, therefore, more imperative than ever that the otolaryngologist should closely follow such patients, since the classical text book pictures of the symptomatology and the pathological findings are so greatly modified that he alone is qualified to evaluate these changes and to act accordingly. The indications for surgical intervention in sinusitis, otitis media and mastoiditis should be recognized, as a recrudescence will undoubtedly come about in the wake of the inevitable limitations to the uses of the antibiotics in otolaryngologic conditions.

SUMMARY.

1. The antibiotics aureomycin, chloromycetin, terramycin and penicillin as adjuncts in the otolaryngologic treatment of diseases of the ear, nose and throat, are discussed in relation to their indications, contraindications and undesirable toxic side reactions.

2. A rapid cultural method is presented for the determination of the sensitivities of the entire flora of the ears, nose and throat for these antibiotics, as a guide to their earlier, more accurate administration.

3. The case is presented stressing the importance of closer observation by the otolaryngologist of patients receiving antibiotic therapy, in the treatment of pathological conditions in his field.

CONCLUSIONS.

1. The antibiotics cause certain undesirable side reactions in human beings: these may be definite contra-indications to their administrations.

2. Different varieties of bacteria are showing greater resistance to antibiotics, which render their administration futile in certain instances.

3. The 24 hours cultural sensitivity tests appear to be 95 per cent as accurate as the 72- to 96-hour methods, as indicators for the administration of the antibiotics.

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A CASE OF ATYPICAL OR QUESTIONABLE MASTOIDITIS IN A THREE-MONTH-OLD INFANT.*

LAWRENCE K. GUNDRUM, M.D.,
UROS A. STAMBUK, M.D., and
JACK W. GAINES, M. D.,
Los Angeles, Calif.

G. D., a three-month-old infant, was admitted to the Pediatric Department of the Queen of the Angels Hospital, April 27, 1953. Mother stated that the patient had had a lump behind the left ear since birth and that it was gradually increasing in size. For the past two weeks the baby had been restless and was not taking food well.

Past History: Full term, normal delivery, normal weight until two weeks previously. *Family History:* Unimportant.

Examination: Showed no pathology in heart or lungs, large swelling in region of left mastoid. Neither membrana tympani could be visualized. White blood count, 19,000. Temperature, 101°F.

Impression: Bony tumor left parietal bone. Seen by the Department of Otolaryngology, April 29, 1953, marked swelling over left mastoid area, softness in superior portion. Right ear normal, left membrana tympani red, with tiny central perforation.

X-ray Examination: Showed soft tissue swelling, no fracture of the skull and no bony involvement. Aspiration into soft area in superior portion of mastoid gave only blood.

Culture: Showed no growth. In view of the leucocytosis, the history, the appearance of the membrana tympani and the condition of the patient, it was decided to perform an exploratory mastoidectomy. This was done April 30, 1953.

* From the Department of Otolaryngology of the Santa Rita Clinic of the Queen of Angels Hospital.

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Soft tissues were found edematous. On removing the cortex, pure blood was encountered. Most of the mastoid cells were gone. Mastoid antrum was located. Wound was closed and a small rubber drain left in antrum.

Post-operative course was uneventful. The drain was removed on the third day; culture after 72 hours showed non-hemolytic-staphylococcus aureus; no recurrence of the tumor, and the growth and development of the child has been normal. The perforation in the left membrana tympani closed.

Comment: Whether or not this was a true case of mastoiditis it is difficult to say. Among other conditions considered was a hematoma due to birth injury, but a normal delivery, without the use of instruments, and the fact that the blood was under the cortex, seemed to rule out such a probability. A diagnosis of hemangioma in such a young infant seemed improbable. With progress some involvement of the middle ear and mastoid now seems indicated.

The Alexander Graham Bell Association for the Deaf (formerly The Volta Speech Association for the Deaf) will hold its 1954 Summer meeting in St. Louis, June 14-18 inclusive. The theme of this meeting is "Let's Face the Issues," and some of the questions to be discussed are: "Where should the deaf child receive his education?" "What are the possibilities and limitations of auditory training?" "What are the principal issues in teaching speech?" and "What contributions can be made to the understanding of the deaf child by persons in peripheral fields?"

All sessions will be held at the Chase Hotel, but there will be Open House at Central Institute for the Deaf and St. Joseph Institute for the Deaf.

For further information, address: Robert Goldstein, 818 S. Kingshighway, St. Louis 10, Missouri.

IMPROVED MODIFICATION OF RIGHT ANGLE SAW FOR RHINOPLASTY.*

ALBERT P. SELTZER, M.D.,
Philadelphia, Pa.

The right angle saw commonly used in narrowing the bridge of the nose, on which a modification is to be described, includes a heavy handle (Lawle's handle), molded to fit the gripping hand with thumb upward, in order to favor the pressure necessary for cutting bone. This saw was designed to aid in fracturing the anterior maxillary processes; therefore, a strong handle is needed.

The overall length of the saw handle is 4½ in. (10.5 cm.), tapering irregularly toward what represents the lower end, when it is gripped, and is formed to turn at about a right angle and extend forward 1¼ in. (2.8 cm.) to the point where the shank of the saw is fixed. The overall length of the saw arm is 3⅛ in. (7.8 cm.), made of stainless steel. The shank of the saw, which is fastened directly into the handle, is bent laterally at an obtuse angle, which brings the saw edge roughly ¾ in. (1 cm.) to the right of the shank by which it is connected with the handle. At the point of the bend of the saw shank, the toothed portion is dropped downward about ⅛ in. (4 mm.) but remains parallel to the shank. The saw itself is 1½ in. (3.8 cm.) long, finely toothed in the ordinary crosscut manner, with the sides of the points equal.

The modification relates to three particulars:

a. The modified handle is made about ½ in. (1.3 cm.) shorter than the usual saw, and the saw shank is fastened directly into the lower end of the handle, extending in the same general direction. An important part of the modification is that, instead of a right angle being formed between the handle and the saw, a moderate acuteness of this angle is produced by having the saw shank set directly in the base

* From the Department of Otolaryngology—Graduate School, University of Pennsylvania.

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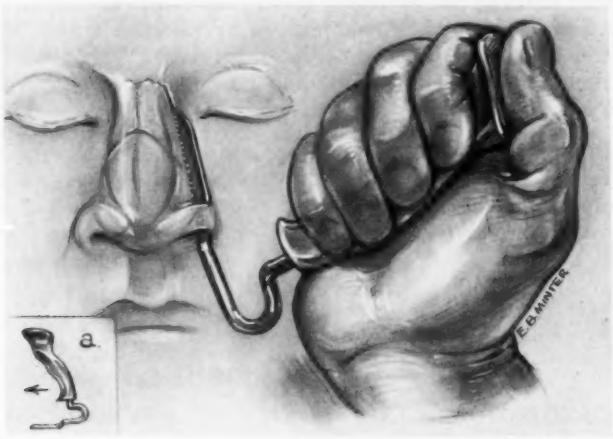
of the handle and angulating the shank, instead of the lower part of the handle itself (see Fig. 1-a). This change of the angle between the handle and the saw increases the potential pressure of the saw upon the bone to be cut.



Proper position of Improved Right Angle Saw.

b. About ⅜ in. (1 cm.) from its insertion into the end of the handle, the shank of the saw is bent to form a U (see Fig. 1-b), which measures ⅝ in. (1.7 cm.) at its open extremity and is ½ inch (1.3 cm.) in depth. This is significant, because it makes it possible for the saw to be easily inserted well up toward the inner canthus, by allowing the overlying tissues to lie in the U-curve, instead of being displaced by upward pressure such as is the case with the other saw.

c. The third detail of the modification is immediately concerned with the cutting edge of the saw, which is of the same size as the original saw, but is slightly thicker. Also, instead of all of the teeth being cut with equal sides, the first five at the tip of the saw are cut so that they point forward, one side of each tooth being longer than the other (see Fig. 1-c), so that when the to-and-fro is made, the effect of the saw is

increased on the bony surface of the anterior maxillary process in the region of the inner canthus. In this way the operation is made easier and a more exact and complete fracture can be effected.

To use the saw, an incision is made with a No. 11 Bard-Parker knife at the pyriform crest, just within the naris, and the tissues are undermined with a periosteal elevator. The undermining here should be extensive enough to allow the entrance of the modified saw and its penetration upward to the inner canthus.

Although the electric saw may in general be more useful, there are times when it may not be available; some choose handpower, either by preference or because they are not sufficiently familiar with the electric saw to use it successfully. Such operators will find this modification more fitting and more suited to their purpose.

2104 Spruce Street.

POSTGRADUATE COURSE IN OTOLARYNGOLOGY

The Department of Postgraduate Medicine of the University of Michigan Medical School announces the Otolaryngology Conference to be given at the University Hospital, Ann Arbor, Michigan, April 15-17, under the direction of Dr. A. C. Furstenberg, Chairman of the Department of Otolaryngology, University of Michigan Medical School.

Guest Lecturers: Dr. J. A. Hilger, St. Paul, Minn.; Dr. H. P. Schenck, Philadelphia; Dr. Paul Holinger, Chicago; Dr. K. M. Day, Pittsburgh; and Dr. Lawrence R. Boies, Minneapolis, Minn.

Resident Lecturers: Dr. A. C. Furstenberg, Dr. J. H. Maxwell, Dr. J. E. Magielski, Dr. Thomas Francis, Dr. Jerome Conn, Dr. E. R. Harrell, Jr., Dr. R. B. Sweet, Dr. H. E. Sloan, Jr., and Dr. Irving Blatt.

For further information, address: Dr. H. H. Cummings, Chairman, Department of Postgraduate Medicine, University Hospital, Ann Arbor, Michigan.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY.

The Sixth International Congress of Otolaryngology will be held in the United States of America in the Summer of 1957. The exact dates and the place of meeting are, at this writing, still undecided. It will be the first International Otolaryngological Congress to be held outside Europe and will afford a welcome opportunity to American otolaryngologists to meet and exchange views with their colleagues from all over the world.

The first of the present series of Congresses met in Copenhagen in 1928 under the presidency of Prof. Schmiegelow; it was attended by 600 members representing 41 countries. The second was held four years later in Madrid, under the presidency of Prof. Tapia; the third in Berlin in 1936, under the presidency of Prof. von Eicken.

At the meeting in Berlin, Holland was designated to take the Congress in 1940 and England, tentatively, in 1944; however, the war intervened, the Berlin records were lost, and further plans remained in abeyance until 1947. At that time, the Dutch committee having indicated its temporary inability to undertake a meeting, the British Association of Otolaryngologists agreed to be hosts to the Fourth Congress, which took place in London in 1949 under the presidency of Mr. V. E. Negus, and was attended by some 1300 members from 45 countries. The Dutch then resumed their obligation and the Fifth Congress was held in Amsterdam, in June 1953, Prof. Eelco Huizinga presiding. The Sixth, to be held in this country in 1957, will be under the presidency of Dr. Arthur Proetz.

Three official themes usually dominate the scientific programs; while the presentations under these heads are by invited speakers, any member of the Congress is eligible to present a paper on a subject of his own choice, and there are sec-

tional meetings in order to accommodate all of the speakers within the time available.

The Congresses have been extremely successful; scientific contributions are many and varied; new friendships are established and old friendships renewed. They have established a sound basis of mutual esteem and understanding by bringing together representatives of countries throughout the world for personal discussion of mutual problems.

Aside from the scientific sessions, social and cultural programs have been arranged which have included specially conducted tours to points of interest in the host city and country. Following some of the Congresses, arrangements have been made for visits to other parts of the host countries for additional special programs. These were both scientific and social in character, and were very successful additions to the Congresses.

PAUL H. HOLINGER, M.D., Secretary-General.

Dr. Sam H. Sanders, Jr., has been named head of the Department of Otology, Laryngology, and Rhinology at the University of Tennessee College of Medicine, succeeding Dr. Charles Blassingame, who asked to be relieved of his administration duties.

Besides his membership in the local and state societies, he is a Fellow of the American Academy of Ophthalmology and Otolaryngology; The American Laryngological, Rhinological and Otological Society; The International Broncho-Esophagological Society; The American Society of Ophthalmologic and Otolaryngologic Allergy, as well as The American College of Allergists; The American Academy of Allergists, and the International Association of Allergists.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

March 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Classic Model T9; Microtone Model T10; Microtone Model T612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor, and 2 tubes.

Manufacturer: Dictograph Products., Inc., 95-25 149th St., Jamaica 35, N. Y.

Audivox, Model 71; 3 transistors.

Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Maico Transist-Ear, Model O; 3 transistors.

Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis, 1.

Otarion Model C-15; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Sonotone Model 1010; 1 transistor, 2 tubes.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes, and 2 batteries (A & B).

Manufacturer: Telex, Inc., Telex Park, St. Paul, 1.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Vice-President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Statler Hotel, Boston, Mass., May 23-24, 1954.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Gordon F. Harkness, Davenport, Iowa.
First Vice-President: Claude C. Cody, Houston, Tex.
Second Vice-President: Daniel S. Cunning, New York, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Meeting: Statler Hotel, Boston, Mass., afternoon, May 27; all day, May 28.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. LeRoy A. Schall, 243 Charles St., Boston, Mass.
President-Elect: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Statler Hotel, Boston, Mass., May 25-27, 1954. (Mornings only.)

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Dean Lierle, Iowa City, Iowa.
Vice-Chairman: Dr. Fred W. Dixon, Rose Bldg., Cleveland, Ohio.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3, Tenn.
Meeting: San Francisco, Calif., June 21-25, 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago 11, Ill.
President-Elect: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Statler Hotel, Boston, Mass., May 17-22, 1954.
Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting: Statler Hotel, Boston, Mass. (Afternoons) May 25-26, 1954.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. Victor Alfaro.
Vice-Chairman: Dr. Irvin Feldman.
Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
President-Elect: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.
Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.
Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. Robert Black, 401 Medical Arts Bldg., Winnipeg, Manitoba.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Harrison Hot Springs Spa, Harrison Hot Springs, B. C.
Time: June 13-15, 1954.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretario del Exterior: Dr. Juan Manuel Tato.
Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
Sub-Secretario Tesorero: Dr. José D. Suberviola.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

SOCIEDAD NACIONAL DE CIRUGIA OF CUBA.

Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho. Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

Presidente: Dr. Aldo Remorino.
Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

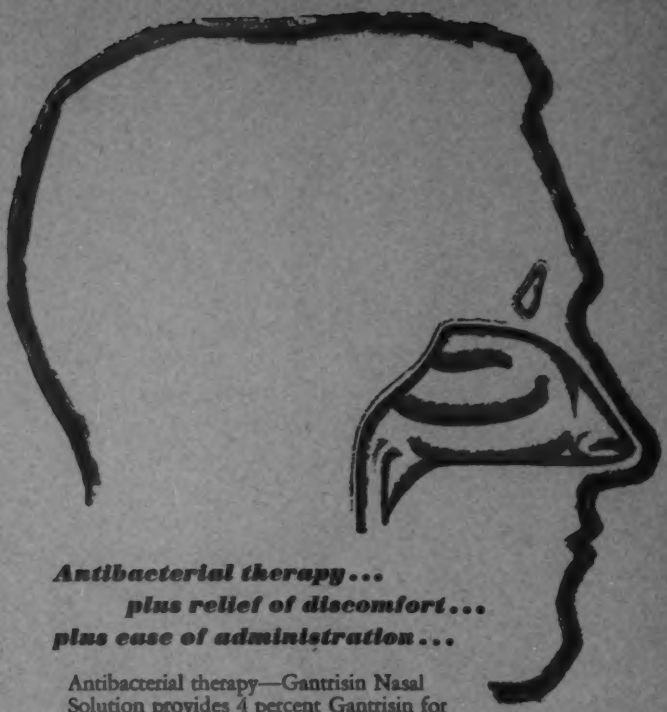
Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: U. S. A., 1957.



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